UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF NORTH CAROLINA
ASHEVILLE DIVISION

STATE OF NORTH CAROLINA ex rel. Roy Cooper, Attorney General,	)	
Plaintiff,	)	No. 1:06-CV-20
vs.	)	VOLUME 4B (Pages 893-1018)
TENNESSEE VALLEY AUTHORITY,	)	
Defendant.	)	

TRANSCRIPT OF TRIAL PROCEEDINGS
BEFORE THE HONORABLE LACY H. THORNBURG
UNITED STATES DISTRICT COURT JUDGE
JULY 17th, 2008

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I N D E X

I N D E X O F E X H I B I T S

THURSDAY AFTERNOON, JULY 17, 2008

THE COURT: Mr. Gulick.

MR. GULICK: Thank you, Your Honor. First I'd like to approach the clerk and deliver the certified copy of the CD of that little film clip that you saw for Your Honor's notebook.

THE COURT: All right. Call your next witness.

MR. GULICK: Your Honor, we call to the witness stand next Dr. David Peden.

DAVID B. PEDEN,

being first duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

BY MR. GULICK:

Q. Would you please state your full name for the record.

A. David Blaine Peden.

Q. And Dr. Peden, where do you live?

A. I live in Chapel Hill, North Carolina.

Q. And where are you currently employed?

A. The University of North Carolina, Chapel Hill.

Q. And what is your current position at the University of North Carolina at Chapel Hill?

A. I have three administrative positions. The first is I'm Associate Chair for Research for the Department of Pediatrics. The second is I'm the Chief of the Division of Allergy, Immunology, Rheumatology and Infectious Diseases. The third

is I direct the research center that the university has, the Center for Environmental Medicine, Asthma and Lung Biology.

Q. And if you could tell us a little bit about your higher educational background, Dr. Peden. First, I guess, starting with your undergraduate courses and degrees.

A. I have an under -- a Bachelor of Arts Degree in Biology that I received at West Virginia University. I stayed there and received a Master's Degree in Pharmacology and Toxicology while I was in medical school where I received my MD. I did my pediatrics residency at West Virginia University. Following that I was an allergy immunology fellow at the National Institute of Health in Bethesda, Maryland.

Q. And following -- following that, following your -- was that your residency?

A. Well, my residency was in pediatrics in West Virginia and then my fellowship from 1987 to 92 was at Bethesda at the NIH.

Q. And if you could, could you please describe your -- the teaching positions that you hold at the University of North Carolina School of Medicine.

A. Well, my -- I'm a professor of pediatrics medicine, microbiology, immunology and toxicology.

Q. And do you hold any adjunct professorships?

A. I hold an adjunct position in epidemiology in the School of Public Health at UNC.

Q. Are you a practicing physician?

A. I am a practicing physician. I direct the allergy and immunology program for my department at UNC.
Q. Do you hold any professional certifications and licenses?
A. I'm licensed in the State of North Carolina. I am certified in pediatrics in allergy and clinical immunology and I'm certified in diagnostic laboratory immunology.
Q. Are you involved in any professional societies?
A. I am involved in several professional societies, including the American Academy of Allergy, Asthma and Immunology, the American Thoracic Society. I'm also past chair and current member of the American Board of Allergy and Immunology which is the agency that writes the examinations to certify allergists and immunologists as board certified. I am also a member of the American Pediatrics -- or the Society for Pediatric Research. And I have others listed in my CV.
Q. Are you a member of the Society of Toxicology or did you say that?
A. I did not say that. I have been a member of the society of toxicology.
Q. Are you involved in any medical or medically related journals?
A. I'm associate editor for the Journal of Allergy and Clinical Immunology. I also review for a number of journals, including the Annals for the American College of Allergy and Immunology. I also review for the American Journal for

Respiratory and Critical Care Medicine, Allergy. I was recently approved as a reviewer for the New England Journal of Medicine. I have also reviewed for a number of toxicology journals in about, I think, 15 that are listed in my CV.

Q. I'd like to go back to your -- you said you were the Director of the Center of -- for Environmental Medicine, Asthma and Lung Biology. Did I get the title right?

A. That's correct.

Q. How long have you been involved in that center?

A. I was recruited to UNC to play a role in that center in 1992. I became the director of that center in 2002.

Q. Could you describe the work for that center for the court.

A. That center was developed at roughly the same time as the Environmental Protection Agency developed a human studies facility on the campus of the University of North Carolina, and we are the university's partner with them in examining the health effects of human volunteers.

And specifically what we do, in essence, is human toxicology. We do human challenge studies so that we can better identify the physiology and the inflammatory effects, the systemic effects of air pollutants in a variety of individuals, although we usually focus on normal, healthy volunteers. We focus on asthmatics. We have studied people with COPD. And we study people across a wide variety of age

ranges.
Q. And when you're doing these, I think you called them human challenge studies?
A. Uh-huh.
Q. Could you describe what's involved in the human challenge study.
A. Well, a human challenge study, first of all, we -- we have to decide what the question is that we wish to address. The chambers that we have access to allows us to study gas phase pollutants, the most common of which we study are ozone. We also have a particle exposure chambers which basically concentrate outdoor particles and vent them into an exposure chamber so that we can do very controlled exposures with particulate. And actually, we can fractionate those on the basis of particle size, including fine mode particles and coarse mode particles.
Q. Would fine mode particles be the same thing as what's been called fine particulates or $PM_{2.5}$?
A. That's correct.
Q. And then how do you go about conducting those chambers studies?
A. Well, again, first of all, we decide what the question is, which population we wish to study. We then -- you know, we being, you know, either myself or others that work with me and usually this is a team approach. We identify which

endpoints we wish to study. Endpoints being of a given pollutant, whether we wish to look at cardiovascular effects, whether we look at pulmonary function effects, whether we want to determine if a pollutant enhances or increases one's response to something they're allergic to. We define what the question is.

Then as a team we develop the experimental protocol. Usually there is one person on the protocol who is the principal investigator. That person is the -- is the overall person in charge of that particular protocol and then there are a number of coinvestigators that either have, you know, nearly coprincipal investigator status or are assigned to the protocol for very specific reasons.

So for instance, if we were going to do a study on the effect of fine mode particles in healthy adults, we would likely have a principal investigator who would be in charge of devising the overall study, developing the study design, developing the statistical approach to the question, which fortunately for us is usually very straightforward.

We also do -- we will also have other members of the team which may include a cardiologist, a pulmonologist if we need to do bronchoalveolar lavage. We will also have other members of the team that monitor the volunteer while they're in the exposure chamber for a defined period of time. We have biostatisticians that work with us. We have an entire

complement of nursing and technical staff, all of whom are engaged in the conduct of a particular study.

Q. Now, are you, as the director, are you involved in design studies?

A. I'm the principal investigator of a large fraction of the studies that our center and our collaboration with the EPA conduct. In addition, as center director, I, with my counterpart in the EPA facility, meet on a monthly basis to review which studies we need to do to make decisions as to what questions or what concerns we need to address, what data we need to generate. And then we will prioritize which studies we do, and then I am either directly involved in planning a particular study or I'm involved in identifying the team that will oversee that study.

Q. As part -- now, you mentioned EPA. What kind of involvement do you have with the Environmental Protection Agency?

A. Well, the -- the EPA -- you know, our facility and our relationship with the EPA has gone back for roughly 30 years. The EPA located its human studies facility on the UNC campus so that they would be able to have a study facility that was on a medical campus. The university helped EPA -- you know, built a building actually for the EPA that's secured on the basis of a long-term EPA lease. My laboratory is within the building. That particular building is actually operated as a

federal facility. And so in a sense I'm UNC's ambassador to the EPA with regards to these kinds of studies, and our job is to really complement what the EPA does. We provide the majority of the medical expertise.

Q. Remember to keep it slow. A little slower.

A. So we, you know, so that's the base -- our long-term relationship with EPA is formulated on the basis of both an institutional memoranda of understanding and we also have a series of cooperative agreements. The cooperative agreement is a funding vehicle that codifies the relationship we have with EPA. I'm the principal investigator or the person in charge of that cooperative agreement.

Q. So you indicated that you're -- are you involved in -- are you involved right now as principal investigator in any studies?

A. I'm the principal investigator of approximately ten studies going on at the present time. I'm a coinvestigator in an additional ten.

Q. And what kinds of -- what kinds of things are you studying at this time?

A. We are currently engaged in a variety of studies. One of our studies is to examine the effect of -- actually, to compare the effect of ozone in the same individual with the effect to endotoxin which is a component of particulate air pollutants.

We're also challenging a number of people to ozone. And once we get enough people, we're actually looking for, you know, for genetic causes to see if there is genetic reasons why some people are more susceptible to the effect of ozone than others.

We are also engaged in studies using our particle concentrators to examine the effect of both concentrated air particulates in diesel exhaust particles or diesel exhaust, actually, in asthmatics and in normal volunteers. We're also examining these effects based on both fine and large mode particles.

Q. And what kinds of endpoints are you evaluating them for?

A. In the majority of our studies we -- in airway chamber study we measure lung function, both because it's of scientific interest and it's also a safety measurement parameter. If we need to make sure that nobody -- you know, if people are beginning to have an immediately bad effect as a result of our study, we can terminate that particular study for that person.

We frequently, not in every study, but we frequently either have them produce sputum so we can get a sense of airway inflammation or they undergo a bronchoalveolar lavage so we can recover airway fluids and study those for both inflammatory cells and inflammatory mediators.

We also on the large majority of our studies now have

Holter monitoring which we use to recover and measure heart rate variability.

And we also collect peripheral blood samples at various time points after and during the study so we can get a sense of what changes have happened immunologically and on an inflammatory basis.

Q. And sort of in layman's terms, what are you looking for?

A. Well, we are looking for the effects of air pollutants both on lung inflammation and lung health that derives in large part from our interest in asthma. And in the normal volunteers and asthmatics, we're also interested in how air pollutants impact cardiovascular endpoints, including heart rhythm or heart rate variability endpoints, systemic inflammatory endpoints. And in some of our studies -- many of these are still underway so we don't have results -- we're looking at vascular reactivity.

MR. GULICK: Your Honor, at this time I would like to tender Dr. David Peden, MD, as an expert in environmental medicine, asthma, toxicology and the biological effects of air pollution on human health, including mechanisms of adverse cardiovascular and respiratory outcomes.

MR. LANCASTER: And Your Honor, we, as I indicated to Mr. Gulick, I believe yesterday, the defendant will stipulate to most of that. That is an awful lot of ground to cover. The portion that we do not stipulate as to which

Dr. Peden should be allowed to give expert testimony is adverse cardiovascular endpoints.

Dr. Peden is obviously a very well qualified doctor in a lot of fields. Just listening to the list of them makes me tired. He is a toxicologist, a pharmacologist, a pediatrician, an asthma and lung doctor, an allergy and immunology specialist, and he is certainly -- we don't stipulate -- object to his qualifications in those fields. He is not a heart doctor. He does work on teams that investigate heart endpoints with other doctors, but he did not disclose any specific training, residency program or anything like that that would give him expertise in that area. He is not a cardio -- he's not a cardiologist.

I know this isn't a binding decision on this court, but it's a sensible one by Judge Voorhees we've cited in some of our briefs called *Smith versus Wyeth-Ayerst*, A-y-e-r-s-t, *Labs*, 278 F.Supp.2d 684, that holds that any expert, including physicians, must have the specialized knowledge or skill in the specific area in which the testimony is proffered. And our position is that asking Dr. Peden to reach out to cardiovascular endpoints as well as the respiratory one extends too far, and so we object only to that portion of his qualifications, cardiovascular.

MR. GULICK: Your Honor, Dr. Peden has just described in considerable detail that he is the director of a

center that works closely with the EPA to study the -- to do research in the very field studying the human health effects of exposure to air pollution, including both fine particles and ozone. And that they -- that they do this to study both the pulmonary effects and the cardiovascular effects, and you've heard that testimony here. I believe --

THE COURT: I don't think he has to be a cardiologist to understand the effects of pollution on the heart --

MR. GULICK: We agree, Your Honor.

THE COURT: -- and related systems. So I will accept the doctor as an expert in the fields as stated and give the defendant the exception as to the field of cardiology.

MR. LANCASTER: Thank you, Your Honor.

THE COURT: Thank you, gentlemen.

MR. GULICK: Thank you, Your Honor.

BY MR. GULICK:

Q. Dr. Peden, when did you first become involved in this case?

A. I first -- it seems like a while ago, but my recollection is in 2006 when your office approached me about my opinions as to the facts and the issues involved in this case with regards to air pollutant effects on human health.

Q. And did you have occasion to generate any expert reports

in this case?
A. I have generated an initial expert report and then a supplemental report subsequent to that, yes.
Q. And one thing I forgot to do, Dr. Peden. I'd like to draw your attention to -- I'm sorry, about this -- your CV which is doc -- which is Exhibit 428. I'd just like to draw that to your attention. It's now before you on the screen and ask you if you can identify that.
A. That is my CV.
Q. And that's Plaintiff's Exhibit 428.
MR. GULICK: Your Honor, I'd like to approach the witness and show him his two reports and ask him to identify them.
THE COURT: All right, sir.
Q. Dr. Peden, I'm showing you what's been marked as Plaintiff's Exhibit 467 and ask you if you can identify that.
A. This is my expert opinion report of October 25th, 2006.
Q. And Plaintiff's Exhibit 468?
A. That's my supplemental report of April 19th, 2007.
Q. Thank you.
MR. GULICK: Madam Clerk, these are for His Honor.
Your Honor, I'd like to move the admission of Dr. Peden's CV, which is Plaintiff's Exhibit 428.
And without belaboring the previous discussions we've had with regard to expert reports, we would also tender

his expert reports, 468 and 467.

MR. LANCASTER: And I have no objection other than the one that has already been noted.

THE COURT: All right, sir. Let those exhibits be admitted.

(Plaintiff's Exhibits Numbers 428, 467 and 468 were received into evidence.)

BY MR. GULICK:

Q. Dr. Peden, do you recall in general what opinions we asked you if you had with regard to the effects of ozone exposure and fine particulate exposure on human health?

A. Well, you asked me to render an opinion as to, indeed, whether ozone and particulate air pollutants have impacts on human health with specific -- you know, we specifically looked at the effects in normal volunteers. We've also looked at the -- we also comment on the effects in asthmatics and on cardiovascular disease.

Q. What I would like to do -- we're going to go into some detail, but what I want to do is to ask you first whether you had and gave us any opinions with regard to the impact of fine particulate matter on cardiovascular endpoints.

A. I think it's very clear that particulate air pollutants have a very deleterious effect on cardiovascular endpoints. They're associated with premature death. They're associated with myocardial infarction, and other cardiovascular, you

know, vascular disease. They're also associated with physiologic changes that are consistent with those, including changes in heart rate variability and changes in systemic vascular inflammation.

Q. We'll go back over those.

And I want to also ask you whether you rendered any opinion with regard to the health effects of exposure to fine particulates on respiratory health.

A. I did. Fine particulate air pollutants have an effect on respiratory health. They are involved in both chronic lung disease and development of chronic lung impact. Acutely they're also implicated and cause asthma exacerbations and exacerbations of other respiratory diseases, including respiratory tract infection and occurrences of acute exacerbations of chronic bronchitis.

Q. And do they have a causal connection with chronic bronchitis?

A. Chronic exposure to increased levels of particulate air pollutants is associated and does have a causal effect with development of chronic bronchitis.

Q. And with respect to exposure to ozone, do you -- did you provide us an opinion with regard to the health effects of exposure to ozone?

A. I have. Ozone is associated with -- ozone actually has two principal effects in humans. One of those is to, I think,

induce an immediate sense of pain, immediate difficulty in taking a deep breath. We've demonstrated that repeatedly in ozone challenge studies. It's also involved with increased airway inflammation. Those are linked in terms of specific diseases. Ozone is associated with decreased respiratory function in otherwise normal, healthy volunteers, and it's most specifically cited to be the cause of increases in asthma exacerbation.

Q. Dr. Peden, I'd like now to go back to the issue of the impact of fine particulates on cardiovascular health. And I'd like to show you for -- marked for identification Plaintiff's Exhibit 194 which should appear on your screen, and ask you if you're familiar with this particular exhibit.

A. I'm familiar with this. This is an overview slide of a presentation actually generated by my colleague, Dr. Devlin, and one that we've used as well in many of our presentations.

Q. And would this particular schematic assist in explaining cardiovascular responses to ambient particulate matter?

A. It will, yes.

Q. So Dr. Peden, would you explain to the court what the mechanisms of cardiovascular responses are to ambient particulate matter.

A. I will. I'll start with just starting at the top, see if I can use this board. Starting out with particulate air pollutants, there are then two ways that PM can have an impact

on cardiovascular disease. One of those is to induce reflexes in the respiratory tract which can translate to increased and modifications of reflex in autonomic nervous systems function. The autonomic nervous system is kind of the rheostat of heart rate and it's actually one of the ways in which the body controls heart rhythm and the speed of heart rhythm.

Q. Bear with us a moment. I think the judge is looking for this particular exhibit.

MR. GULICK: I'm sorry, Your Honor. It's Exhibit 194 and it should be in Notebook Number 3. I apologize.

THE COURT: Notebook 2.

MR. GULICK: Did I say 2 or 3? I meant 3.

Did you find it?

THE COURT: I do have it now.

MR. GULICK: I apologize.

THE COURT: That's all right. I didn't have it before.

All right. We have it now so go right ahead.

MR. GULICK: My apologies.

I think we might back up because I think Your Honor was -- I apologize, Your Honor. I'm still a little bit -- I've never used this electronic myself.

Q. Dr. Peden, could you start again with your explanation with regard to this particular table.

A. Certainly. After exposure to particulate air pollutants,

there is a -- you know, there can be one of two pathways that are activated after exposure in the respiratory tract. The first I will talk about is activation of both pulmonary reflexes which then act -- which induce activation of the autonomic nervous system. That's the sympathetic nervous system which is the kind of the fight or flight reflex. That's what -- you know, that's the epinephrin-based reflex system. And the one that controls that and down regulates that is the parasympathetic nervous system, and those two things in balance represent the major function of the autonomic nervous system and those things help keep the heart rate in appropriate balance.

When the autonomic nervous system is modified, there's a change in conduction and repolarization of the heart. What I mean by that is that heart rhythm, the electrical activity of a heart which basically coordinates how a heartbeat occurs, involves the SA node activating and transmitting signals to the AV node That then goes through basically the Purkinje Fibers which is the wiring of the heart, and then with that is a coordinated depolarization, an electrical switch of every cell -- of every heart cell that then allows that cell to contract. If the heart contracts in a coordinated fashion, you get good blood flow throughout the body. If the heart cells are contracting de-synchronously, that's ventricular fibrillation and left, you know, unchecked for more than a

couple of minutes, that leads to death. And there are variations of heart arythmias in between that that occur.

By affecting the autonomic nervous system, one can affect heart rate and cardiac rhythm and with that you'll have this, you know, malignant arythmia, and that's basically what I just spoke to.

You've heard me refer a couple times to this notion of heart rate variability. That is a measure that is used and that literally is that. If one looks at the -- at the heart rate or the R to R interval, that's the -- if you looked at EKG's, the big spike on the EKG, the really big spike that goes up, that's the R wave and the R to R interval. That's also in heart rate variability parlance known as the end to end space.

Q. Just keep your pace down.

A. That -- in general, and particularly in people who have had previous cardiovascular injury, they either have had a myocardial infarction or they've had congestive heart failure. What can happen if there's a difficulty in the heart to respond to these autonomic nervous system signals, the heart is unable to respond to environmental stress and that is a risk factor for sudden death. And that's -- actually, inability to have or decreased heart rate variability or the decreased ability of the heart to either change or increase or lower its rate in response to environmental stress is

associated with increased risk for sudden death or arythmia.

The reason I point that out is that the vast majority of, particularly panel studies -- and I'll get into what an epidemiological study is, what a panel study is and then my expertise which is what a chambers study is. But in panel studies, and to a certain extent in chambers studies, what have been found, particularly in elderly populations, increased exposure to $PM_{2.5}$ is associated with decreased heart rate variability. So this PM signal and its effect on the autonomic nervous system reactivity is associated with a change that previously, you know, before this phenomenon was really being studied in the context of air pollution had been associated with increased risk for cardiac death.

Q. Thank you.

A. So moving from there, I'll point out that the autonomic nervous system is also involved in blood pressure control. And blood pressure, particularly transient increases in blood pressure -- increased blood pressure can actually be a problem because the heart has to work harder, particularly if your diastolic or your resting blood pressure is increased, the heart has to work harder to get blood into the tissues and that's one reason why blood pressure control has been such an important issue. Without the ability to keep your blood pressure normal, that puts strain on the heart. And for a person who's at risk for heart disease, either because they

have preexisting coronary artery disease or had previous heart injury, that stress will be quite significant.

The blood -- you know, the -- working against the pressure head will be significant. And in fact, PM has been associated particularly in elderly populations with at least modest, and also in a controlled exposure study, PM has been shown to be modest -- modestly increase diastolic blood pressure.

Moving away from the reflex side of this, I would go more towards the pulmonary inflammatory system. And this is where -- this is where actually my specialty does bleed in, pardon the pun a little bit, into cardiology because these effects are actually systemic inflammatory effects which are very much the parlance of what I'm clinically trained to deal with which is systemic inflammation.

And what happens when systemic inflammation impacts the cardiovascular system, there are a couple of things that happen. One is that there's endothelial cell and vascular dysfunction. The endothelial cells are the cells that line arterioles and arteries, but in there there are molecules, particularly nitric oxide, that's involved in being able to dynamically respond to environmental stress. And vascular reactivitiy may be affected and there are studies that demonstrate that with air pollutant exposure, vascular contractility is such that there is less blood flow. And so

that can happen as a result of particulate air pollutant exposure.

Lastly, to be -- try to be to the point with this, the systemic inflammation can also affect platelet function and it can also activate clotting factors such as von Willebrand Factors

Q. You might need to spell that.

A. Thank you. V-o-n -- it's a name, small letter V, W-i-l-l-e-b-r-a-n-d. Named after a German physiologist.

And this clotting factor -- and there are other systemic inflammatory factors that have been associated, including C-reactor protein which is a very nondescript -- you know, when I say nondescript, it's a very general sense of increased inflammation generated, in fact, by the liver, but it has become a very cardinal mark for systemic inflammation and increases in C-reactive protein are now used by both family practice docs who follow people with cardiovascular disease as well as cardiologists to get a sense of what the risks for vascular aspects of this would be.

And so there's an increase in C-reactor protein associated with PM exposure. There are changes in fibrenogen which is also part of the clotting factor and modifications of the --

THE COURT REPORTER: I'm sorry, I didn't understand that. Fibrenogen which is also part of the clotting factor and

modifications...

THE WITNESS: Fibrenogen is one of the -- is another one of the clotting factors involved in forming a clot. So if you have a sudden clotting event, that would cause a myocardial infarction or stroke.

When these inflammatory effects occur and if someone has preexisting atherosclerotic plaque, it's thought that there could be plaque rupture that would lead to thrombosis and ultimately either stroke if it happens in your brain or heart attack if it happens in the heart.

The things that have been really well documented in humans -- I mean, we've never done a study and to my knowledge we've not done studies where we've observed -- we don't intentionally put people in PM chambers to give them strokes. But what we do do is take relatively, you know, at safe exposures and safe paradigms, we study this and we actually study the effect on clotting factors. We study the effect on heart rate variability and we've been able to see effects on systemic and pulmonary inflammation. And all those things have been seen both in panel studies and also in chamber studies associated with PM.

Q. And that forms the basis of your opinion.

A. This paradigm and the observations that lead to this paradigm contribute to the basis for my opinion that PM is causative for cardiovascular disease.

Q. And that -- and the range of these cardiovascular diseases include what?
A. One can have subacute, subclinical vascular inflammation that increases risk for causes of death due to other coincident causes all the way to sudden death, and occurrences of acute tachy or abnormal heart rhythms.
Q. So could this also lead to lesser cardiovascular events such as hospitalizations?
A. Yes. I'm sorry, I misunderstood. One -- it's associated with death, admission to hospital. Subsequent to admission to hospital, you know, there's lost work time. And depending upon the degree of injury, there's chronic -- there can be chronic disease. So a person who's had a myocardial infarction can then go on and have chronic congestive heart failure which impacts their activities of daily living.
Q. Do you have any further things that you would like to say at this stage about the impacts of fine particulate on cardiovascular disease?
A. I mean, I think that we've covered the one -- that fine particulates are associated with acute exacerbations of disease. They're aassociated with all the events we've just spoken to. And the majority of the data are associated with those. There are certain risk groups and people in certain risk groups that are at higher risk for that. In addition to those who have had previous cardiovascular disease, there are

studies implicating diabetics having increased risk to any cardiovascular event, including those that are associated with particulate air pollutants.

People with a metabolic syndrome, which is kind of a prediabetic state usually associated with certain degrees of low grade systemic inflammation, vascular inflammation and overweight, those folks may likely be -- there's evidence that they're also at increased risk for cardiovascular disease associated with PM.

Q. Does this mean that if somebody were to have a sudden cardiac death as a result of this, that they were about to die anyway?

A. Well, certainly if they were about to die anyway, it can; but most of the data don't support that. Most of the data do not support that you have basically early reaping of people who are otherwise, you know, clinging to the fringe of life. These are people who otherwise would have been perfectly well and could have had an undefined life-span, but they die as a result of the air pollutant event.

Q. Doctor, could you describe for us in a bit more detail what the impacts of ozone exposure are on human beings.

A. Well, on an epidemiological scale on a population basis -- I'll speak first to the diseases that are associated with it and then I'll speak to what the human exposure challenge studies do that document how we understand, you

know, to the extent that we understand how ozone injures or affects individuals.

Increased ozone exposure even at levels below the current NAAQS standard has been associated with -- the disease that's been probably the most strongly associated with is asthma. There's very little debate, frankly, or there's no substantial debate that increases in ozone exposure are associated with increases in asthma exacerbations. Typically, the pattern for that is that it's usually 24 to 48 hours after the increased ozone, there will be a subsequent increase in hospitalizations due to that effect.

Q. Why is that?

A. Why is that? Well, that gets -- we believe why that is is that there -- and what we know from our challenge studies is that acute exposure to ozone will increase acute inflammation, inflammatory cells present in the airway. And inflammation is a cardinal feature of asthma, including neutrophils which are the immediate inflammatory cell. And neutrophils are actually increased in the acute phase of an asthma event as well.

There is also increases in the kinds of cells called monocytes and macrophages, and those cells are engaged in -- can be engaged in control of inflammation.

All of those things are increased, and we believe that what happens is that the increase of those cells increase the

responsiveness in an allergic asthmatic to something that they're already allergic to. What we have definitively shown in our studies and others -- and there's probably ten such studies if not more across a variety of ozone exposures that demonstrate that exposure to ozone will increase one's responsiveness to allergens.

So the way we've done these studies is we do a placebo control, so we expose somebody to clean filtered air in a randomized blinded fashion, you know, so they get placebo, they get clean filtered air, they get exposed to ozone. After the ozone exposure and after the placebo control, we have them undergo a graded inhalation challenge with different doses of allergen until they have a very small but noticeable drop in lung function. That's the typical endpoint. That allergen mimics an asthma attack.

And the vast majority of those studies have demonstrated that there is increased reactivity to allergen after ozone response. And I and I believe most of my allergy/immunology colleagues who study this are confident that one of the ways that ozone affects asthma is to increase one's response to something that they're allergic to. There are a lot of data that support that.

In addition to that particular effect. There's a completely separate effect of ozone that's not engaged with asthma, that's not an asthma effect, although this could

affect asthmatics. And this was defined in the mid '80s through the mid '90s across an entire dose response of ozone. And it turns out that people acutely, not a day later, but acutely will have a pain response that's mediated by C-fibers and mediated by pain receptors in the lung and that response can go on at the same time as the inflammation goes on, but the two are really not the same. So you can have lots of pain response with not a lot of inflammation or vice versa.

But what we know is that humans will have an immediate acute phase response to ozone which is a pain response. Experimentally, if we give them anti-inflammatory drugs -- or let me rephrase that. When we give them drugs like Ibuprofen, you know, that you would use for a headache, you know, we can give it to prevent lung ache as well. In other words, if you give that prior to the ozone challenge, one will mitigate that effect. And if you give narcotic analgesics experimentally during that -- you know, during the course of that, you will completely reverse the restrictive lung effect. And what I mean by restrictive lung effect is that -- you know, this pain response means you can't take a really big deep breath. That therefore limits the amount of air that you can get in, and as that continues the dyspnea or the feeling of shortness of breath and the feeling of lung discomfort really exacerbates. That will go on for probably anywhere from one to three hours in most individuals after stopping the ozone challenge.

The dose of ozone that causes this effect has been studied and really the total dose of ozone is a function of how fast you're breathing. So in other words, minute ventilation, the duration of exposure and the level of ozone present, all three of those factors go into calculating the total dose.

So for instance, a person exposed to a very high level of ozone but who's at rest and is not breathing very rapidly and therefore -- and is exposed for a relatively brief period of time, even though they're in a high ozone level for that moment in time, their total ozone dose is going to be very abbreviated. Conversely, if a person who might be exposed to a fairly modest level of ozone compared to the previous one but they were undergoing exercise and at the same time were exposed for anywhere from two to four hours -- and in our experiments we have done experiments looking for as little as one hour and as long as 6.6 hours. That's how you fit in an eight hour work day with a couple of breaks for the staff. You know, looking at those kinds of exposures, that, you know, we find, you know, that some people will have a response, a lung function response.

We're currently engaged -- there's been a re-analysis of a study by Adams looking at .06 part per million ozone. That re-analysis by EPA has led to actually a new study being done that we're in the midst of to confirm that that level of ozone

will cause immediate phase lung responses. So even at levels below the current standard, there's concern that that immediate phase response will happen.

It's also very clear that there's person-to-person and very likely genetic variability within that nonasthma acute phase response to ozone. Some people are exquisitely sensitive. Some people will do an ozone challenge and very shortly after the exposure begins -- we're planning a two to four hour exposure, after, you know, 30 to 60 minutes we have to abort the exposure because their lung function has dropped, you know, by 40 percent and they're feeling very uncomfortable and we have to stop the exposure. Some people are much more tolerant of the ozone effect. And that's one of the reasons we're doing studies now is that we're trying to determine genetic markers.

But these are people who are not asthmatic; who, by every physical parameter we've looked at when we screen them for study, are not identified as being different. They don't have a disease. They're usually between the ages of 18 and 30. And within those populations you see this sort of variability. But there's a fraction, you know, a significant fraction of people that are remarkably sensitive to the effect of ozone.

Q. Dr. Peden, were you present during the testimony of Mr. Will Harlan?

A. I was.

Q. And you heard all of his testimony.
A. I did.
Q. Do you have any opinion with respect to what was occurring in his situation?
MR. LANCASTER: Your Honor, I would simply interpose an objection that this opinion was not included in the expert disclosures that Dr. Peden made.
MR. GULICK: Your Honor, I don't think Dr. Peden had heard the circumstances until he heard this testimony.
THE COURT: All right. I'll let him answer. Overruled.
A. The description that we heard is extremely consistent with the description and the observations we make with volunteers that undergo an ozone challenge in our studies, and we've probably literally -- over ten years we've probably challenged four or five hundred people at least in the variety of studies we've done. And his description is absolutely typical of what people describe. They describe an inability to take a deep breath and a tight pain in the chest and a substernal discomfort. And left alone when they come out of the chamber, because we're not in the habit of giving narcotic analgesics routinely unless we have a very defined experiment to do that. Within a couple of hours that usually subsides and by the next morning they're usually -- you know, we've had one or two exceptions, but typically people -- you know, that

effect goes away fairly quickly.
Q. Was there any significance to the fact that he was exercising?
A. Well, as I just said, the dose -- the effective dose for an individual with an ozone exposure is a function of minute ventilation, how fast you're breathing and how deeply you're breathing. In most of our ozone challenges, we try to effect a minute ventilation of around 30 -- 25 to 30 liters a minute which is -- represents a typically fit -- we can argue what typically fit means, but a typically fit individual walking at about three and a half miles an hour, intermittent exercise and rest so that on average there's a 25 liter per minute respiratory rate. Most normal adult humans will breathe somewhere between 8 to 10 liters a minute at rest. A trained athlete will be -- I can't guess what his minute ventilation was other than to make a rough guess that it was significantly above 25 liters per minute. We've seen that when we have athletes really push themselves. So his minute ventilation was higher -- I suspect higher and he was running all day. So, you know, assuming that the ozone levels were -- you know, you have that evidence. He had increased exposure -- duration of exposure and he had increased minute ventilation.
Q. Thank you. Doctor, are there other effects from exposure to asthma that you -- I'm sorry, pardon me -- exposure to ozone that you have described to us in your opinion?

A. I have. most -- many of these derive from epidemiological studies and professional colleagues of mine -- you know, again, our forte has been, what we do in our facility are panel studies, you know, studying a small number of people intensely with natural exposures and the chambers studies. But there's a robust body of evidence that demonstrates that exposure to ozone may be associated with causation of asthma. This was a study done, and I believe this is cited in my expert opinion as one of the references. A study done in southern California looking at high level ozone areas and low level ozone areas examining children as they entered -- I've forgotten off the top of my head the age, but I think -- I want to say at fifth grade and then again at eighth grade. And they looked at the children who were not identified as having asthma and then those that subsequently were newly identified as having asthma and they looked at the relative risk of factors associated with that. And the risk factors that were very clear indicators of risk were living in a chronically high ozone area within southern California. Believe it or not, southern California is not ubiquitously highly polluted. There are pockets of relatively clean air and pockets of very, very highly polluted air. And those that live in the highly polluted areas that practiced outdoor aerobic sports were -- that was the marker for minute ventilation. They were the ones that were about 3.3 fold

higher likely than those who didn't develop disease to have disease. That is an example of ozone being associated with causation of disease.

There's also data that demonstrate that people who live, you know, during their childhood in a high ozone area, many of these derived from the University of California system looking at freshmen. Those who live in a highly polluted area will have lower lung function, not drastically lower, but lower lung function than those who live in chronically less polluted areas.

Q. What does lower lung function mean?

A. Well, lower lung function, what I mean by that is the forced vital capacity which is a measure -- the amount of air -- you know, if you go to an allergen or pulmonary specialist, they will have you, to look at lung function, blow into a device and they'll coach you -- they'll actually scream at you and yell at you, but they'll try to get you to blow out as much air as you possibly can. That total amount of air that you can voluntarily get out is called forced vital capacity. The amount of that air that you can get out in one second is called the forced expiratory volume at one second.

The forced vital capacity is the principal point and that is lower, statistically significantly lower in people who live in higher pollution areas. The same effects on lung growth have also been shown with NOx or oxides of nitrogen. It's the

Gauderman Study in the New England Journal, 2004. I think it's in the 2004 New England Journal.

Q. Dr. Peden, I think I skipped over the effects of fine particulate on respiratory disease -- of exposure to fine particulate on respiratory disease. Could you discuss that.

A. Well, again, epidemiological studies have demonstrated that there is a clear link between increases in PM_{10} -- or I'm, sorry $PM_{2.5}$ and occurrences of asthma exacerbation and the timing is just relatively similar to the effect -- to the effect on -- of ozone, and we believe that represents a PM_{10} induced inflammation that then renders you more susceptible to things that one's sensitive to with regards to asthma.

We have specifically studied components of $PM_{2.5}$. The components my laboratory studied is endotoxin which is found in both coarse and fine mode particles. And like ozone we find that those materials enhance response to something that one is allergic to.

We also know from PM from -- CAPS particle exposures when we do bronchoscopy -- this is -- most of these have been done by my EPA colleagues. But when CAPS or Concentrated Air Particulate studies have been done followed with bronchoscopy to get airway samples, there's an increase in inflammation in normal -- you know, most of these have been done in normal healthy people, but there's an inflammatory effect with those as well.

Q. And how does that relate to -- what respiratory diseases does that relate to?

A. Well, chronically that can be associated with chronic bronchitis. There's an association with acute changes -- PM exposure in increase respiratory tract infections. Perhaps the best epidemiological study that comes immediately to my mind is the Utah Valley study in which there was a kind of, you know, in the late '80s where there was kind of an economic experiment. There was a steel mill and there was a steel mill strike. And in the years flanking the strike, there was both a spike in PM exposure and associated with that were marked increases -- or -- and this happened typically in January -- increases in admission to hospital for a variety of respiratory diagnoses, specifically bronchitis, asthma and some pneumonia type diagnoses. The year of the strike when nothing else hypothetically had really changed in the Utah Valley, there was a notable decrease in the wintertime particulates and coincident with that there was a marked decrease in the incidents of all those diseases. When the strike was resolved and the plant resumed operation, all of those events came -- you know, were recovered.

Those kinds of events and the association of PM with those sorts of exacerbations have been shown in literally almost too numerous to count studies.

Q. Dr. Peden, have you read the expert reports of

Drs. Moolgavkar and Anderson that were submitted on behalf of the defendants in this case?
A. I have read those reports.
Q. Is there anything in their expert reports that have caused you to change any of the opinions that you have rendered?
A. There is nothing in those reports that cause me to change my opinions. I think Dr. Moolgavkar's opinions are -- represent a minority view in -- with regards to the majority of the air pollution scientists community. And I understand the points that he makes, but the preponderance of the evidence with regards to particulate air pollutant effects on either mortality or other disease exacerbations, in my view, clearly demonstrates a relationship and a causative effect of PM on a variety of health outcomes.
Q. Does that include premature death?
A. It does.
Q. Drs. Moolgavkar and Anderson have suggested that we've not demonstrated biological plausibility of those effects. Do you agree with that?
A. I do not agree with that. In my view, biological -- biological plausibility implies both the epidemiological studies -- which, again, I don't agree with those. I'm not a epidemiologist and you'll have other testimony for that. But also in animal systems and in human challenge systems we very

clearly demonstrate that exposure to -- experimental exposure to particulate air pollutants, experimental exposure to ozone clearly cauesd a biological --

COURT REPORTER: I'm sorry, you lost me.

THE WITNESS: With regards to experimental evidence, you know, in addition to epidemiological evidence, animal data, human challenge data, clearly demonstrate the capability of particulate air pollutants, ozone, to exert effects in biological systems, including intact human beings. They cause inflammation. They cause systemic inflammation. Can cause changes in heart rate variability.

And so the idea that air pollutants cannot have an effect on human health systems is -- you know, I don't hold that view. I disagree with Moolgavkar on that point.

Q. Do you think that their view is shared by the majority of knowledgeable scientists in this field?

A. I do not believe that. I believe that their view is not shared by the majority of the scientific community that studies this.

Q. Dr. Anderson has suggested that there should not be any harms to exposure to human -- to human beings by either fine particulates or ozone below the current national ambient standard. Do you agree with that?

A. I do not.

Q. And why not?

A. I do not because there are -- you know, there are a number of epidemiological studies that demonstrate just in the area that -- with regards to asthma exacerbation, again, clearly published that levels as low as .05 and .06 can be associated with exacerbation of asthma and with regards to ozone.

There are -- and there are studies that demonstrate that low level exposures to PM -- most of the studies associate this with a 10 microgram per cubic meter increase and those studies demonstrate increases in cardiovascular effects.

So in essence, my opinion is that there -- that even levels of pollutants, and certainly of ozone and PM, below the current NAAQS still exert deleterious effects and cause exacerbations of disease.

MR. LANCASTER: Your Honor, I rise only to note an objection to Dr. Peden relying on studies without identifying them.

MR. GULICK: Your Honor, I think they'll have an opportunity to cross examine him.

THE COURT: Just a minute.

You'll have an opportunity on cross to seek that information.

MR. LANCASTER: Thank you, Your Honor.

Q. Dr. Peden, are there biological reasons why you would -- that would support your view that there would not be a

threshold at the NAAQS?
A. Well, we've -- there is -- in all the -- in human exposure studies, and including the low level studies, there are always -- there have frequently been a handful of people that will respond to -- just acutely respond to ozone even at levels below the NAAQS. Because of the number of people that one can study in a human chamber study, it's difficult to demonstrate statistical significance because you have to enrich those studies with a large number of studies that look at these people. But there's always -- there is always a handful of people that will respond to levels below the NAAQS level, and indeed, to get at with regards to ozone. Specifically, the re-analysis of the data of the .06 level and in our current study -- you know, that current study is current so we don't have results yet -- is designed to get at specifically that point.
Q. Does that opinion also apply to exposure to fine particulates?
A. It does.
Q. Dr. Peden, in one of your -- you attached an article to one of your -- to your second -- to your supplemental report authored by two gentlemen named Pope and Dockery.
A. That's correct.
Q. And do you recall -- if you'll bear with me a moment.
Do you have that there?

A. Yes, I do have the article.
Q. The bottom -- this is -- if you would go to page 732 of that article which is attached to your appendix as Appendix 1 to your supplemental opinion dated April 29 -- April 19, 2007.
The journal article is page 732 in the bottom left-hand corner.
A. Okay.
Q. And there is a sentence that begins about -- if you have a hard copy, about an inch and a half up that begins -- following a quotation, it says, "A comprehensive evaluation of the literature." Do you see where I'm talking about?
A. I do.
Q. Could you -- could you read that sentence aloud and then tell us whether or not you agree with it.
A. Okay.
MR. LANCASTER: Your Honor, I would like to note an objection to this. Dr. Pope and Dr. Dockery who wrote this article are not expert witnesses in this case and we object to nonwitnesses in the case being attempted to be introduced through other witnesses and would request that Dr. Peden state his own opinions.
MR. GULICK: I'm going to ask him if he agrees with what's stated there, Your Honor. That's all.
THE COURT: All right. You may proceed.
THE WITNESS: Okay. "A comprehensive evaluation of

the literature provides compelling evidence that continued reductions in exposure to combustion-related fine particulate air pollution as indicated by $PM_{2.5}$ will result in improvements in cardiopulmonary health."

Q. Do you agree with that statement?

A. I do agree with that statement.

Q. In your opinion, does that -- does that represent the majority view of knowledgeable scientists in your field?

A. It does.

Q. In summary, Dr. Peden, is it your opinion that there is a causal relationship to an exposure of human beings to ozone in the ambient area and respiratory hospitalizations?

A. There is.

Q. Do you have the same -- is it your opinion that there's a causal relationship between exposure of humans to ozone in the ambient area and asthma exacerbations?

A. There is a causal effect.

Q. Is there a causal relationship between exposure of humans to ozone in the ambient area and asthma emergency room visits?

A. There is.

Q. Is there a causal relationship between exposure of humans to ozone in the ambient area and lost school days?

A. There is.

Q. In your opinion, is there a causal relationship between exposure of humans to fine particles in the ambient air and

premature death?
A. There is.
Q. Is it your opinion -- in your opinion, is there a causal relationship -- let me start again.
In your opinion, is there a causal relationship between exposure of humans to fine particles in the ambient air and cardiac hospitalizations?
A. There is a relationship.
Q. In your opinion, is there a causal relationship between exposure of humans to fine particles in the ambient air and respiratory hospitalizations?
A. Yes.
Q. In your opinion, is there a causal relationship between exposure of humans to fine particles in the ambient air and asthma emergency room visits?
A. Yes, there is.
MR. LANCASTER: Your Honor, I just note an objection to the leading of Mr. Gulick's own witness.
MR. GULICK: Your Honor, I'm just summarizing his previous testimony.
MR. LANCASTER: In that case it's cumulative, Your Honor.
THE COURT: Objection is overruled.
MR. GULICK: I'm close to the end, Your Honor.
Q. In your opinion, doctor, is there a causal relationship

between exposure of humans to fine particles in the ambient air and asthma exacerbation?
A. Yes.
Q. In your opinion, is there a causal relationship between exposure of humans to fine particles in the ambient air and chronic bronchitis?
A. There is.
Q. In your opinion, is there a causal relationship between exposure of humans to fine particles in the ambient air and lost school days?
A. There is.
Q. And finally, in your opinion, is there a causal relationship to exposure of humans to fine particles in the air and minor restricted activity days?
A. There is.

MR. GULICK: I have no further questions, Your Honor.

Your Honor, I did have another exhibit which was Exhibit 194 which I'd like to -- which was that diagram which I'd like to offer for illustrative purposes.

MR. LANCASTER: No objection only for illustrative purposes, but objection for substantive purposes. The witness has testified that someone else prepared it.

THE COURT: It's admitted for illustrative purposes.

(Plaintiff's Exhibit Number 194 was received into

evidence.)

All right. Who proposes to cross examine Dr. Peden?

MR. LANCASTER: I'll have that pleasure, Your Honor.

THE COURT: All right.

CROSS EXAMINATION

BY MR. LANCASTER:

Q. Dr. Peden.

A. Yes, sir.

Q. For North Carolina's adults, isn't it true that asthma prevalence has remained below the national median for the last six years?

A. That's likely true.

Q. In fact, North Carolina has consistently had a lower current and lifetime asthma prevalence than the national median; isn't that correct?

A. That's correct.

Q. Less than about 7 percent of North Carolina's adults currently have asthma; isn't that correct?

A. That's correct.

Q. Isn't it true that North Carolina has had a significant decrease in mortality rates due to asthma since the mid 1990s?

A. That's true.

Q. And asthma is a treatable disease, is it not?

A. Generally, yes.

Q. And it can be controlled by treatment.

A. Yes.
Q. Would you agree that hospitalizations due to asthma are often preventable and result from uncontrolled asthma which could be avoided with good asthma management techniques?
A. I would agree with that.
Q. Likewise, emergency room visits for asthma are often preventable.
A. They are.
Q. In terms of hospitalizations for asthma, isn't it true that asthma hospitalization rates in North Carolina are generally higher in the eastern part of the state than in the western part of the state?
A. That may be. I don't know directly to answer that question.
Q. Are you familiar with a document published by the North Carolina Department of Public Health called the Burden of Asthma in North Carolina 2006?
A. I am -- I haven't -- I haven't -- I don't have an encyclopedia. I'm aware of the document, yes.
Q. And if you would take Book 16 from the shelf behind you, please. The cart I should say.
A. Book 16. Okay.
Q. We may have reference to that in a moment. I want to ask you this. Is mortality due to a primary cause of asthma, is that a relatively rare event?

A. It is a rare event.
Q. And like emergency room visits and hospitalization, deaths due to asthma commonly represent a breakdown in successful disease management.
A. That is true.
Q. Isn't it true that indoor allergens and irritants often play a significant role in the triggering of asthma episodes and attacks?
A. That can be true, yes.
Q. And secondhand smoke is one example of an indoor asthma trigger, isn't it?
A. It is.
Q. Secondhand smoke can trigger asthma episodes and increase the severity of attacks.
A. Yes.
Q. And isn't it true that more than 40 percent of North Carolina's middle schoolers and high schoolers live in homes with smokers?
A. That's probably true.
Q. Isn't it true that more than a fourth of North Carolina's high school students are themselves smokers?
A. I don't know. I'll -- I was going to use the word stipulate, but that's one of your terms.
Q. Cardiovascular disease death rates have also been declining in North Carolina; isn't that correct?

A. That's correct.
Q. In North Carolina, higher cardiovascular disease death rates are clustered primarily in eastern North Carolina, correct?
A. That's likely true.
Q. And likewise, cardiovascular disease hospitalization rates are clustered primarily in eastern North Carolina; is that correct?
A. Yes.
Q. And the highest heart disease death rates are clustered in eastern North Carolina; is that correct?
A. That's correct.
Q. And the highest stroke death rates and hospitalization rates are clustered primarily in the coastal plain region in eastern North Carolina, correct?
A. That's correct.
Q. If you would turn to Exhibit 408 in the book that you retrieved from the cart.
A. Okay.
Q. And what is Exhibit 408?
A. The Burden of Asthma in North Carolina 2006.
Q. And is it a publication of the North Carolina Division of Public Health of the North Carolina Department of Health and Human Services?
A. It is.

Q. If you would turn, please, to Page 69.
And does Page 69 give an indication of asthma hospitalization rates in North Carolina from 2002 to 2004?
A. It does.
Q. And does it appear to indicate that the highest hospitalization rates are occurring in the eastern part of North Carolina?
A. Generally, yes.
Q. And so does that refresh your recollection that in fact the highest asthma hospitalization rates occur in eastern North Carolina?
A. Yes.
Q. Now, in your testimony you've mentioned epidemiological studies showing association between exposure to pollution and health endpoints; is that correct?
A. That's correct.
Q. Epidemiology is a science that's used to examine the pattern of disease in human populations; is that correct?
A. Correct.
Q. Epidemiology is largely an observational discipline, isn't it?
A. That is generally true, yes.
Q. That is, epidemiological studies are not based on clinical trials or experimental studies where subjects are randomly assigned to groups in which one is exposed to a

pollutant of concern and the other group is not, correct?
A. Oftentimes that's true. There are exceptions to that. There are -- for instance, there's a genre of epidemiological research known as panel studies where you don't change the experimental exposure to the pollutant, but you can certainly randomize people to treatments or the things that might mitigate the effect of pollutants or not.
Q. Are you familiar with an epidemiological study known as the American Cancer Society study?
A. Yes.
Q. Is it true that the American Cancer Society study shows a negative statistically significant association between fine particle levels and death caused by diseases of the respiratory system?
A. As I recall, that's true.
Q. And what that means is that the American Cancer Society study is showing that when fine particulate -- excuse me, what the American Cancer Society study is showing is that when fine particulate matter levels go up, deaths are going down; is that correct?
A. In that study, deaths to the respiratory tract reasons were going down.
Q. Right, I'm sorry, yes, for respiratory deaths.
And the American Cancer Society study is likewise showing that when fine particulate matter levels go down, respiratory

deaths go up; is that correct?
A. In that steady that relationship was seen, yes.
Q. Now, when an epidemiological study does find a positive association between an exposure and an outcome such as death, that is not by itself equivalent to causation, is it?
A. Generally not. I mean, the cause -- you know, the relationship is demonstrated and then there are both animal and human studies that will look at, you know, try to develop or go after specific biological mechanisms. The preponderance of epidemiological studies shows a very strong relationship between particulate air pollutant increases and respiratory and cardiovascular death.
Q. Would you agree that no matter how strong the association between exposure and disease as shown by an epidemiological study, it is difficult to accept the association as causal when no mechanism can be identified by which the exposure leads to the putative effect?
A. When there is no evidence for a mechanism, it's more difficult, I will agree with that.
Q. And you yourself acknowledge, don't you, sir, that the underlying mechanisms for the effects of pollutants are incompletely understood?
A. I do accept that.
Q. And you read some lines from the Pope and Dockery 2006 article; is that correct?

A. That's correct.
Q. And in your expert report, you indicated that you had attached the Pope and Dockery article and stated, "Their overall assessment, which is consistent with my own, is that 'despite important gaps in scientific knowledge and continued reasons for some scepticism, a comprehensive evaluation of the research findings provides persuasive evidence that exposure to fine particulate air pollution has adverse effects on cardiopulmonary health.'" Is that correct?
A. That's correct.
Q. So you acknowledge that there are continued reasons for some scepticism.
A. I acknowledge that there are -- there are stated reasons for some scepticism.
Q. And you acknowledge that there are important gaps in scientific knowledge.
A. There are important gaps in scientific knowledge.
Q. Drs. Pope and Dockery in that 2006 article also stated, "There remains a need for a healthy scepticism regarding what we may think we know about the health effects of particulate matter exposure." Do you agree with that statement in the article?
A. I'll agree with that statement.
Q. Now, you indicated a reference to numerous studies. Can you identify any studies that associate or relate in any way

to determining whether exposure to nitrate particles in amounts no greater than 0.3 micrograms per cubic meter causes mortality?

A. I can't immediately recollect that off the top of my head, no.

Q. Can you identify any studies in which exposure to sulfate particles at levels no greater than 0.3 micrograms per cubic meter cause mortality?

A. I can't specifically remember the number. I can't remember -- there are articles that I've read since these reports were made that relate sulfate to morbidity. But specifically answering your question, I cannot off the top of my head, no.

Q. Are any of the articles that you -- or studies or research or experience upon which you have relied for support for your conclusions, do any of those address the question of whether exposure to fine particulate matter at levels no greater than 0.3 micrograms per cubic meter could cause mortality?

A. Not in my recollection, no.

MR. LANCASTER: I have no further questions, Your Honor.

THE COURT: Redirect.

REDIRECT EXAMINATION

BY MR. GULICK:

Q. Dr. Peden, does the list of statistics about North Carolina that was being recited change any of the opinions that you've rendered in this case?
A. They do not. There are multiple reasons why the burden of disease in eastern North Carolina can be greater than in other areas, you know, not simply -- not only the environmental issues. So the short answer is no, it doesn't change my opinion. There are clearly data that demonstrate that even in relatively healthy populations where there's less of a disease burden, but within those populations when there are increases in pollutant exposures, exacerbations of disease occur in those regions.
Q. Doctor, are you aware of any reason why exposure to the sulfate particle component of fine particulates should be exonerated from the rest of the fraction of fine particulates?
A. Well, I mean, I -- no. There are data that suggest, you know, in one of our studies that I believe is actually in our expert report, we looked at fractions of particles that included, you know, iron, sulfate and selenium, and in those fractions, those particles that were -- they were in highest concentration, it was the most significant increase in inflammation in the normal, healthy bronchial study as an example. But I'm not aware of studies that exonerate sulfate as an important contributor.
Q. Just to go back to that particular study that you were

mentioning. Could you restate what that study showed in that particular case?

A. Well, this was the CAP study in which -- this is the CAP study in which concentrated air particulates were -- people were exposed to that and because the concentrated CAPS particles, you know, when they're collected, the amount of different components of those were analyzed using mass spectrometry, and sulfates were -- you know, and in the batch that contained the highest amount of clustered materials, including iron, sulfate and selenium, that's where -- it's in that fraction where one also sees the greatest amount of -- in that (cortile quartile), actually, was where one sees the greatest amount of inflammatory response to the CAPS.

Q. Are you aware of any -- doctor, of any basis for finding a threshold below which there is no effect on human health from exposure to either ozone or fine particulate matter?

A. I'm not aware of any clear data that demonstrate a no effect threshold.

MR. GULICK: No further questions.

MR. LANCASTER: May I ask two further questions, Your Honor?

THE COURT: Yes.

RECROSS EXAMINATION

BY MR. LANCASTER:

Q. Dr. Peden, you just mentioned a study, and if you said

the name of it, I apologize, I didn't catch it. What was that -- what study was that?

A. I believe the study is the study by Huang, and it's in my supplemental report.

Q. I guess this will be three questions. Could you spell that name for me.

A. H-a -- H-u-a-n-g.

Q. And what was the concentration exposure level of the subjects in that study?

A. It varied, but it could be up to 200 micrograms per cubic meter.

MR. LANCASTER: Thank you. No further questions, Your Honor.

THE COURT: All right. That will conclude your testimony, then, Dr. Peden. Thank you very much and you may be excused.

THE WITNESS: Thank you.

MR. GULICK: Thank you, Your Honor.

(Witness stepped down.)

MR. GULICK: Your Honor, our next witness is Dr. Donald Russell.

DONALD W. RUSSELL,

being first duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

BY MR. GULICK:

Q. Dr. Russell, could you state your full name, please.
A. Donald W. Russell, MD.
Q. Where do you live, Dr. Russell?
A. South Asheville, North Carolina.
Q. How long have you lived in Asheville?
A. Eighteen years.
Q. You're a medical doctor?
A. I am.
Q. Where do you work in Asheville?
A. I work at what used to be called Mountain Allergy until just recently we changed our name to Allergy Partners because of growth in our group. Mountain Allergy and Asthma Associates, PA, now Allergy Partners. That clinic is in Asheville, but I have several satellite clinics that we travel to one or two days a week.
Q. I'd like to show you on the monitor Plaintiff's Exhibit 441. It should appear before you in a minute.
Do you see that?
A. I do.
Q. And I want to ask you if that is your curriculum vitae?
A. It is.
Q. Dr. Russell, where did you go to medical school?
A. Emory University School of Medicine.
Q. And when did you graduate?
A. 1984.

Q. Where did you do your residency?
A. At the University of South Florida School of Medicine.
Q. And what was the subject of your residency?
A. I did that in internal medicine, in which I'm board certified. Then I did an additional nearly three years in allergy, asthma, immunology subspecialty at the same institution, the University of South Florida, affiliated hospitals which included the large VA hospital in Tampa, Tampa General Hospital, and also All Children's Hospital and Immunology Center in St. Petersburg.

And I'm board certified in allergy, asthma and immunology, just to complete the record.

Q. Thank you. And Dr. -- Dr. Russell, how long have you worked at what was Mountain Allergy and Asthma?
A. Since I came here in 1990.
Q. And your specialty is in allergy and asthma?
A. Allergy, asthma and clinical immunology.
Q. What is clinical immunology?
A. We -- it's an interesting overlap with rheumatology, but mostly in looking at children and adults that have antibody deficiencies, recurrent infections because of immunologic disorders, identification of infants with inherent defects in their immune system, dysfunctional bone marrow, and so on. Often we're the ones responsible for giving kids the old -- what used to be called the old gamma globulin injections. Now

it's usually an infusion, a slow infusion of gamma globulin to replace missing antibodies with folks that identify the specific defects that would lead them not only to a diagnosis of humoral or antibody deficiency, but the treatment for that. In fact, we're the only specialty that does that.

Q. You're a practicing physician?

A. I am indeed.

Q. And do you regularly see patients?

A. I do.

Q. Is that here in your various offices here in Asheville and in satellite towns?

A. Right, in satellite towns: Waynesville and Franklin.

Q. And what kinds of diseases do you primarily treat?

A. Allergic diseases of the respiratory tract is my main focus in the volume of patients. And that includes --

Q. Does that include asthma?

A. It includes asthma to a large percentage.

MR. GULICK: Dr. Russell, if it's necessary -- Your Honor, if it's necessary to do so, Dr. Russell was not specially retained for any purposes, but he has both factual and other knowledge, but clearly he is qualified by training and experience to talk about his treatment of his asthma patients. If it's necessary to do so, I would ask that he be qualified as an expert in allergy and asthma.

MR. LANCASTER: And Your Honor, the defendant has no

objections to Dr. Russell's qualifications and would not question those.

We do have an objection to Dr. Russell providing expert opinion testimony and that objection is solely that no report was provided of Dr. Russell's opinions. In this lawsuit there are about 20 expert witnesses who among them have provided about 50 expert reports, including those we discussed this morning that were provided late. And we believe that the expert testimony in this case should be limited to the experts who actually provided expert reports.

We've cited a couple of cases in our trial brief on this issue. One from the Eleventh Circuit and one from Judge Anderson down in South Carolina involving an airplane crash case, both of which support the propriety of ruling that experts who don't provide reports ought to be excluded even if they are testifying free of charge.

MR. GULICK: Your Honor, Dr. Russell has been identified on our witness list since sometime in 2006. Has been a medical doctor. We provided his curriculum vitae. He was not specially retained by us. We provided a brief on this particular subject, that he would be available to testify about the effects of air pollution on his asthma patients. And the defendants have had notice of that fact since the very beginning, since our very first -- the very first list of potential witnesses that we provided. And they have had

plenty of opportunity to take his deposition.

Indeed, he also since then supplied, in response to a motion for summary judgment, supplied a declaration which was included in our response to the motion for -- their motion for summary judgment.

So the defendants have had adequate notice of Dr. Russell and the potential for his testimony. And we believe that since he was not specially retained, that that notice was sufficient and within the keeping of the rules.

MR. LANCASTER: Your Honor, my memory may not be correct. There may have been more than 50 witnesses -- more than 50 witnesses, potential witnesses on plaintiff's list and we didn't want to have to depose every one to find out what they would say. We believe it was plaintiff's obligation to provide a report of expert opinions if they would be given just as they did for Dr. Peden from whom we just heard, although Dr. Peden was not specially retained either.

MR. GULICK: Your Honor, I simply want to point out that the Federal Rule of Civil Procedure specifically distinguishes between those who are specially retained and others who may have knowledge arising -- and the rule makes clear, I'm referring here to Federal Rule of Civil Procedure 26(a)(2)(B). It says that -- and I'm referring here to the cite of a case from our brief, *Carr versus Dietz*. This is in our brief at Page 2. That the rule imposes additional

requirements if expert witnesses -- if the expert witness is retained or specially employed to provide expert testimony. So there's a different requirement for those experts who aren't specially retained.

THE COURT: All right. I'm going to let him testify. Defendant had full knowledge that he was available to testify -- for testifying and you were already on notice by what the state has furnished in a previous part of preparation for this trial that Dr. Russell could or might be called, though I agree that traditionally you should be notified. And I would expect in the future for either side to be more careful as they prepare for the trial to give opposing counsel notice if they -- of their witness list to give them ample opportunity to do depositions if they choose. There was enough notice there, it seems to me, for defendant to at least make inquiry as to whether this witness would be called. And certainly you have, I'm sure, defense witnesses who are going to be testifying in this same field. And after your cross examination, your witnesses, I take it some of whom are in the courtroom, will have the opportunity to contradict Dr. Russell if they disagree with what he has to say.

So I'll let him testify and give you an exception to that, Mr. Lancaster.

MR. LANCASTER: Thank you, Your Honor.

MR. GULICK: Thank you, Your Honor.

BY MR. GULICK:

Q. Dr. Russell, as Dr. Peden has already testified, can you briefly describe what asthma is for purposes of your testimony.

A. Asthma is a rather -- it's a nuisance disease. In common vernacular in medicine, it's a twitchy airway. What that means is the bronchial tubes, at least as far as the symptoms go of sudden reversible tightening in the airway, those smooth muscles that wrap around or go through the bronchial wall are twitchy. They tighten to a number of factors or stimulants ranging anywhere from cold air, rapid drying of airway, the release of histamine from allergy cells, chemicals from allergy cells also now known as leukotrienes, some of this stimulated by irritants, pollutants, allergens, and several other factors that you heard testimony to today.

But the real underlying disease process is not just this twitchy airway. That's just the phenomenon. That's the tip of the iceberg that we see. That which lies below the water of the lower part of the iceberg, if you will, is inflammation, and it's a very profound inflammation in some cases. What we call our moderate or severe asthmatics have an influx, a residence, if you will, a taking up of a residence of white blood cells infiltrating the bronchial wall that can stay there and be very difficult to remove, and that in turn leads to hypersensitivity of the smooth muscles that I was

talking about that wrap these bronchial tubes, millions of them together in the lung. And any slight decrease in the radius of the bronchial tube increases the resistance to breathing and it increases the effort of breathing, the required effort by the power of four. And that's just by changing the radius.

There's also in many asthmatics --

Q. Doctor, if I might, I'd like to show you what's been marked for -- as Plaintiff's Exhibit 213 which should appear on your screen.

This is Plaintiff's Exhibit 213. And let's wait for His Honor to...

THE COURT: All right. Now I have it.

Q. Dr. Russell, can you tell us -- this is a -- appears to be a model. Can you tell us what it shows.

A. This is a cross-section of the human bronchial tube which appears to be fairly normal in its diameter, the caliber of the opening. Looking at the shadowed area of the tube are conduits through which air travels. There's the gray items in the wall of the bronchial tube that are on cut end are mucous glands. You can see one of them with a conduit for mucous traveling into the bronchial tube. And then the redder snakelike or wormlike projections are smooth muscle cut on end or on foss. And then underneath those or medial to those going toward the opening of the tube are blood vessels. The

little red dots are blood vessels cut on end. And this is a fairly normal, uninflamed bronchial wall, although it could be a mild asthmatic's bronchial tube as well. You may not see much difference on biopsy.

Q. I'd now like to show you what's been marked as Plaintiff's Exhibit 214 and ask you if you can identify what this is or tell us what this is or what it shows.

A. What you see here -- what you see here is the same type of cross-sectional cut of bronchial tube that is showing a significant tightening of the smooth muscles. As you can see, now they're more arcuate. They're also thicker, which is one of the things that increases the reduced diameter, or reduces the flow of air is that in asthmatics, the more times their airways are twitchy, it's like lifting weights or pumping iron, they become thicker. That also increases the thickness of the bronchial wall and thus reduces the amount of conduit for air, and thus increases the resistance to air flow by the power of four.

Then there's also the production of the light colored, yellowish gray mucous which some asthmatics produce. It produces an even greater and very significant reduction in air flow if they are mucous producers. They tend to be our more difficult asthmatics. They tend to follow along with high levels of eosinophils. Some of the cells that Dr. Peden alluded to, monocytes, macrophages, neutrophils, lymphocytes

and eosinophils. And in the -- many of our asthmatics, the majority would have a lot of eosinophils, which are allergy cells, taking up shop or residence in the wall of the bronchial tube in an asthmatic leading to increased inflammation.

And in the mucous glands, notice there are more mucous glands in this cross-sectional biopsy, the gray rings. Those are goblet cells or mucous glands producing excess mucous in this patient which leads to increased expectoration of a white or gray sputum when they're having a flare and sometimes when they're not having a flare. Sometimes ambiently and daily.

Q. When you say a flare, is there another name for that?

A. An asthma attack. Asthma exacerbation.

Q. Doctor, you had described increased resistance when breathing. Is there something that you can -- if -- when there's this constriction, is there a way that you can liken that to something that would describe for those who don't have asthma what it's like?

A. More articulate asthmatics will often say the attack was sudden in onset and it was like breathing -- trying to breathe through a straw. And if it was a severe attack, they would even say -- more articulate, more worldly would say it was like breathing through a martini straw.

Q. And the result of that is -- what does that do for the person who's trying to do that --

A. Well, first of all --
Q. -- breathe like that?
A. -- for very many patients it's frightening. They feel like they may suffocate. Smothering is the most common word I hear from patients here in the mountains. Doc, I'm smothering. And you often know that's an aggravation of their COPD, their chronic lung disease or their asthma or chronic bronchitis or viral or walking pneumonia, perhaps. And sometimes that intense emotional response, the fright or fear, sometimes backfires. You think adrenaline would bronchodilate, but it doesn't always. Sometimes it acts through the parasympathetic system to broncho-constrict.

These people have very twitchy airways and just going down the wrong aisle in the grocery store or going out on a bad air day will often produce increased symptoms and require them to take more medication or alter their activities of daily living.

Q. Doctor, you've heard the testimony of Dr. Peden. But in your experience as a practicing physician in this area, is there effect of high particulate days or ozone days on your patients?
A. It didn't take long, maybe my first year or so in practice, to know that there was a subset of patients who were inordinately sensitive to high -- high levels of haze in the environment. When you couldn't count the trees on the

mountains, they couldn't go out and do their gardening. They couldn't run or jog. They couldn't ride their bikes. And some couldn't go to work. Just going outside for the brief period of time to get to their car or walk from the parking lot to their place of business or employment, they would have a flare-up of asthma. And it often occurred, which was an interesting observation, at times when the pollen counts, which is another major trigger for asthma in our mountain areas at certain times of the year, occurred during the lull periods for pollen, where we've about run out of grass pollen, the trees are essentially all done and all we have are heat and perhaps bad air quality to aggravate or prevent activity, outdoor activity and impair the quality of life of our patients.

Q. You were describing these restrictions on what they could do. Is that what's known as a minor restricted activity day --

A. Well --

Q. -- for those patients?

A. I guess it depends on the individual. You heard Mr. Harlan's testimony. That's a major interruption of his activities. You know, this is part of his life, his running.

But for some, you know, the patients may not complain as much. They're retired. They have other things they can do indoors. If they can afford air conditioning and stay

indoors, that tends to help them. Or they avoid the highest level of irritant time of day, for example, midmorning through later in the afternoon or early evening when the pollutants and the irritants can be seemingly the highest, particularly noted for ozone, the research that we heard testimony on today.

Q. How do you advise your patients how to deal with this, with bad air days?

A. Well, the advice is to stay in on really bad days if you can help it. If you can't, there may be some things that you can do. Our medications, though -- I'll very respectfully disagree with the conclusion by other counsel or Dr. Peden. There are some patients for whom control of their asthma is inadequate no matter what we do, and those are our failures. Those are the folks who end up making their way to the emergency room either because of a virus, high pollen counts, air pollution or exertion or what have you. And those patients, no matter what we do, will have an exacerbation that results in either hospitalization or acute treatment in our office, or emergency room visits most likely. And those ER visits are expensive. They begin at 13 to 15 hundred dollars. And if they come to our office, it's less expensive. We don't charge a hospital fee. But if they get admitted to the hospital, it's a minimum of $5,000 on average. I just saw those figures for our hospital locally.

So there's a subset of patients that are very difficult, but for the vast majority I do agree that our medications do a good job; notwithstanding the fact, though, that they're extremely expensive.

Q. Doctor, I'd like to draw your attention -- could you explain to us what kind of medications are required for the treatment of asthma.

A. We have two treatments for -- historically and for many decades we had the short-acting bronchodilators which relaxed bronchial smooth muscle, and that was when we only saw the tip of the iceberg. But it was, of course, the bottom of the iceberg that sunk the Titanic. And we finally stumbled on, about 25 years ago, the incredible inflammatory part of the disease common with the disease that we were missing. In fact, I remember being a freshman medical student -- or a third-year medical student, actually, on the wards at Grady hearing, well, don't let the asthmatic die without a trial of steroids. Now we use steroids every day and frequently and liberally especially in the inhaled form.

So those inhaled steroids are the other component. Immediate bronchodilators: Albuterol or levalbuterol, immediate, short-acting bronchodilators. And of course, now there are new generation of long-acting bronchodilators that last up to 12 hours.

But the other component, component two is the

anti-inflammatory treatment and this gets -- this is where a lot of the expense comes. These anti-inflammatories are inhaled. They're topically deposited in the lungs. And because they require special technology or dry power technology and special delivery units, there is a lot of research that went into that. There's high cost of production, and unfortunately, high margins of profits required of the patient to pay dearly as well as the payors, Medicaid and private insurance, costing up to 150 to 200 dollars for a one-month unit for the average moderate asthmatic.

Q. On bad air days, when you detected them as being bad air days --

A. I do.

Q. Do you --

A. Excuse me, I thought you were finished with your question.

Q. No. When they occur, do you see -- do you see an influx of more patients after that?

A. We'll often have -- usually a day or so later into it because patients don't always call the minute they start to feel badly. Or it's also thought to be a delay in the onset of inflammation or at least the manifestation of inflammation in the asthmatic patient, at least, from exposure to peak symptoms.

But we'll often get the day -- the afternoon of or the following day or two days later or for that whole period of time an increase in the number of phone calls for refills on inhalers that have long expired by months, six months, nine months. And they'll also ask for their anti-inflammatory medication, especially if they're a well-educated, well-trained asthmatic knowing that they need to follow their asthma action plan whenever their peak flow meter readings drop by 12 to 15 percent or when their symptoms peak, the two criteria we use for gauging when to apply higher doses of the anti-inflammatory and regular doses of the bronchodilator therapy, the fast-acting treatments.

So those are the only -- those are the two major components. There's another anti-inflammatory medication out there called Singulair. There's actually two, Singulair and Zilutin, monoleukast is the generic name, and zileutin, now known as far as proprietor names as Singulair and Zyflo CR. And those, also very expensive medicines, are frequently added in as an anti-inflammatory. Not particularly potent except for a small subset of patients. But often patients do recognize some extra or added protection or relief when those extra -- those other -- that other category of medicines called the anti-leukotriene or leukotriene blockers are added. Another expense.

So between the -- if they're allergic, and many of our

asthma patients are allergic. 50 to 70 percent of our asthmatic patients started out with allergic rhinitis. Another target disease process that appears to be aggravated by air pollution, sinus problems, as well as the allergies in our area. And if you have a person who is allergic and asthmatic, they can be on as many as five or six different medications.

Q. How much does that cost a month?

A. Five hundred to 800 dollars. The range would be 200 to 800 dollars for maybe asthma controlled by a single medicine which is that category of patients who are mild intermittent. But our moderate, persistent and more severe asthmatics will require three to five medications in most cases.

Q. Do you have -- do you treat Medicaid patients?

A. I do.

Q. And approximately what percentage of your patients are Medicaid patients?

A. Approximately 25 percent.

Q. And do they have to deal with the same expense of medication?

A. Not as much as the person with private insurance. But they still have to make small co-pays for either their allergy treatment and sometimes their medication, and they -- but the burden to Medicaid, as well as a few of the Medicare part D programs, is rather -- rather high and extensive when you

think of what the insurance companies are having to pay for these medications.

Q. Doctor, I'd like to show you a document which is Plaintiff's Exhibit 45.

MR. GULICK: And Your Honor, this is an affidavit -- Your Honor, this is an affidavit which is -- we have it from George Carr who is a -- which has been provided to defense quite some time ago. He's a Decision Support -- George Carr is a Decision Support Manager in the Division of Medical Assistance. This is a document in which he authenticates information that he had gathered that he was the custodian of -- it was electronic information about expenditures by the Medicaid program of the State of North Carolina.

I have the original affidavit in my hands and I'm about to hand it up. However, I would like for Dr. -- for Dr. Russell, who is clearly acquainted with the medications and treats Medicaid patients, to identify some of the drugs that are listed by name in this document.

MR. LANCASTER: Your Honor, we have a hearsay objection to this document. It's an affidavit and not -- there's no hearsay exception to admit Mr. Carr's testimony by affidavit with no cross examination in this case. Mr. Carr is clearly under the control of the plaintiff as he is an employee of the State of North Carolina.

MR. GULICK: Your Honor, this affidavit is an

authentication of records of the State of North Carolina and we believe it meets the tests under Rule 803 as a hearsay exception. Specifically, records, reports, statements or data compilations in any form of a public office, agency, setting forth the activities of the office or agency, which his affidavit does, matters which there is a duty to report. And in here it's specifically reporting records of the State of North Carolina and we believe it meets the hearsay exception for such records.

MR. LANCASTER: I believe 803(a) requires that it be prepared in the ordinary course of business and this document was prepared apparently for this litigation. And our position is that it's a hearsay document and that Mr. Carr himself ought to testify as opposed to trying to appear by affidavit.

MR. GULICK: Your Honor, I think he's referring to the regularly conducted activities. What this -- what these are are public records of the expenditures of the Medicaid program.

THE COURT: Let me see the record and the affidavit.

MR. GULICK: The record files and the affidavit, Your Honor.

(The documents were tendered to the court.)

THE COURT: All right. Overruled.

MR. GULICK: Thank you.

THE COURT: We'll -- it's 4 o'clock so I think we'll

take our midafternoon 15 minute break, doctor.

(Brief recess at 4:07 p.m.)

THE COURT: All right, Mr. Gulick.

MR. GULICK: Thank you, Your Honor.

DONALD RUSSELL

DIRECT EXAMINATION (Cont'd.)

BY MR. GULICK:

Q. Dr. Russell, we have in front of you a page which you can't see --

MR. GULICK: Can you blow that up a little bit at the top. Can you expand it at the top.

That's good. Thank you.

Q. Dr. Russell, this is the document the judge has just examined. And what I want you to do is to -- you see under it it has a column that says "Asthma Medication Expenditures."

A. Yes, I see it.

Q. And below that it identifies -- appears to identify the names of what may be drugs. I was wondering if you could identify for us if these are drugs with which you are familiar.

A. Very familiar.

Q. What are these drugs?

A. These drugs are the drugs in my armamentarium of treatments for asthmatics and allergic patients and especially those who overlap with both nasal and sinus allergies as well

as asthma. And asthma is very often an allergic disease of the lung and inflammation that I described having come from -- often from allergy or other proinflammatory processes.

Q. This is a long list. I was wondering if you could identify a few of these as ones that you regularly prescribe and tell us what the cost is.

A. Singulair, $110 a month both for the adult and the pediatric dose which you see both there in that order respectively.

Advair, 140 to 250/50 dose is $145 for only 60 updrafts which would be a one month supply.

The Pulmicort is about $110 per month in the respul form. This is the type you open up, you put it in the bronchial nebulizer, and it's a little plastic tube, you drop the single dose in. Often you take it once to three times a day. That's the pediatric -- or the intermediate pediatric dose. There's a higher dose for adults.

Q. Are any of these rescue medications?

A. What we call -- refer to as the quick-acting or rescue medications. You come down to Xopenex which is --

Q. Does that start with the X?

A. Yeah, it's pronounced Z even though it's an X. X-o-p-e-n-e-x.

Xopenex is a fast-acting medication that's now available in an inhaler form in what we call the HFA preparation,

hydrofluoroalkane preparation. And that replaces the freonated fast-acting or quick-acting medicines which we knew of as simply generic albuterol. And now there's albuterol in the HFA form, nongeneric, which has increased the cost. Albuterol was available as a freonated device. It's being phased out by December. It's my understanding that it will no longer be legal to prescribe or refer a patient to buy albuterol with freon in it because of the protection requirements for the ozone layer.

Q. So is albuterol a rescue inhaler?

A. It is. Xopenex and albuterol are basically identical drugs. One is a more purified mixture of the single effective -- most effective isomer of albuterol. The levalbuterol is Xopenex. It's more expensive, as you can see.

Q. What are these rescue drugs used for?

A. The sudden tightening of the bronchial tubes, and they relax -- they only relax the smooth muscle tightening which I said is the twitchy airway, if you will, that narrows the bronchial tube due to tightening of the smooth muscle coils that embed or surround the bronchial wall.

Q. And when would a patient -- when would you instruct a patient to use such a thing?

A. When they're having a flare-up of their asthma symptoms. When they feel tight. When they're coughing, and some patients all they do is cough. And for others it's when

they're wheezing. For a vast majority of our patients it's when they're wheezing.

Q. And what is wheezing?

A. Wheezing is that musical, dissonant sound that comes out of the lung when many different diameter bronchial tubes are tightening and it causes a sibilant resonance that sounds like dissonant music. Many bagpipes and flutes playing all at once. I can demonstrate if you want to hear. I have asthma.

Q. And is that a common symptom of asthma?

A. Very much so. Probably more so than the cough, though that's debatable. There are a lot of cough variant asthmatics. I can't give you an exact split.

Q. You testified a while ago that after a bad air day, that you would get a call for refills.

A. Yes.

Q. Is that often -- was that for this kind of rescue medication or was it for some other type?

A. It is. In so many of the patients, unless they're a well-trained asthmatic, will call for simply the rescue medicine, the quick-acting bronchial dilatator, forgetting that they also need to stop the inflammation produced by the bad air days. And that's a subset or overlap of subset of patients who have both allergy and a sensitivity to hazy air days.

Q. Doctor, do you know a percentage of your patients who are

affected by bad air days?
A. Within my degree of medical certainty and my clinical experience, it's somewhere around three out of five. It's the majority of patients. It's not small by any means. It will often have them have to take their bronchodilator quick-acting medicine before they even go outside to go to work, to do their outdoor activities, work in the garden, walk the dog.
Q. Doctor, do you know whether or not there's a significant frequency in the population of people with undiagnosed asthma?
A. I can't give you the exact figure, but I did hear a figure from my academy, the American Academy of Allerery, Asthma and Immunology, the AAAAI, that allergists see only 2 to 3 percent of all the patients that should be seen. That they need a specialist. People who frequently report to the emergency room or have uncontrolled symptoms or who have been on a ventilator, who have almost died from asthma, who can't sleep at night because of their asthma, those patients aren't getting to us mostly probably because of underinsured status. They're not insured or they don't have enough insurance to pay for their care or they're not eligible for the government programs.
Q. Doctor, do you have experience with patients who have uncontrolled or poorly controlled asthma?
A. I do. And there's a significant number of them. It's not the vast majority. It is a subset. I can't give you my

exact figure, but it's approximately 1 in 10 to 1 in 20 of my asthma patients, and I can see as many as 40 a week in my practice, asthmatics.

Q. And what can be the consequences of uncontrolled or poorly controlled asthma?

A. The worst consequence, our greatest fear, after hospitalization because then we get a black mark from the insurance company if our patient gets admitted to the hospital. We cost them too much money. But the worst fear is the development of a process called remodeling of the lung. It's a scarring down of the parenchymal part of the lung or the subendothelial tissues in both the bronchus intermedius as well as the smaller bronchials. And that process, if the asthmatic is caught too late, if he started on anti-inflammatory treatment anywhere from two or three years into his asthmatic process, if his primary doctor doesn't start him on an anti-inflammatory or if he's a specialist who probably would too late, that patient can develop scarring of the lung and lose 10, 15, up to 50 or 60 percent of their lung function and never recover any of it.

That was a big study done in the Netherlands, and I can give you the name of that with a little -- this afternoon if you need it tomorrow. But that was a beautiful study. You're looking at Budesonide and they divided the patients according to how long they had symptoms before they were start on

anti-inflammatory treatment.

And so many patients, because of underinsurance, if you'll allow me that expression, won't access the subspecialty care or good primary care soon enough before they lose lung function. And sometimes -- there is a definite subset of patients, and this has been described in the Journal of Allergy and Clinical Immunology over and over again. There's a subset of patients no matter what you do, they do not improve and they continue in some cases to go downhill and lose their lung function and they are what we call pulmonary cripples by the time they are middle age or older. On oxygen sometimes.

Q. Doctor, can asthma be fatal?

A. Absolutely.

Q. Have you ever observed a fatal asthma attack?

A. I did. Fortunately, none of my immediate patients. But when I was a fourth-year medical student at Grady Hospital in Atlanta, a little two-year-old boy whose parents would not stop smoking around him. He lived in the inner city, and there were many factors. And he came in dead on arrival, two-year-old boy, that had just been there just a week earlier and whose parents were warned not to smoke around him. He was put on Prednisone and he died anyway. They fished out -- you saw the mucous in the diagram, the second exhibit with the cross-section of the bronchial tube. That mucous formed,

looked like an oak tree without leaves in the wintertime here in the mountains. They fished two of those out of each lung -- one out of each lung and that -- he had -- he didn't have a prayer to oxygenate.

Q. What happened -- so what happens in that event?

A. Well, you hope that you could keep this child alive. He, of course, died.

Q. What's the cause of death?

A. Asthma.

Q. I mean, but what's the -- why --

A. Oh, respiratory failure. Immediate cause of death respiratory failure, secondary asthma.

Q. Doctor, are you concerned as a result of your experience with your patients, are you concerned about air pollution?

A. I am indeed.

Q. In addition to the treatment of your patients, have you done anything as a result of that to deal with air pollution?

A. Well, I did some militating, if you will, for the Smokestacks Clean Air Act here in North Carolina. I called several of my local representatives. I was in on a -- invited to join and became a member of a planning committee to have an impact on our legislators to try to get scrubbers on our power plants here in western North Carolina and made phone calls and wrote letters.

Q. So you lobbied for the Clean Smokestacks Act?

A. I did. That's the word I was groping for, lobby. I did lobby for that.
Q. Doctor, do you have any -- have you had any other personal, outside from your medical experience, have you had personal experience with -- of a separate nature involving air pollution?
A. Well, for one thing I have asthma. Some of you may have heard me cough a little bit. The walk around town here got me going. And the other is that I'm a pilot. And I'm a pilot of about maybe 11 years' duration now. And I remember taking my check ride some years ago and again more recently, within the last three or four years, having to use instruments to find the airport in daylight hours. It was summer and it was 4:30 to 5:00 p.m. And I remember having the tower told me that I was a mile and a half off course for the inbound line to the runway. And I said, well -- I thought I was looking at the runway; I was looking at I-26. And so they asked me if I needed the rabbit lights to come on, and that's a pilot term. And with air traffic control, rabbit lights are the huge flashing arrow that's about maybe 1200 yards long that directs you to the runway. And I had to have them turn it up to the second level of frequency before I could see it. And I had 20/20 vision at the time.
Q. And why was that?
A. Because the air was so dirty or so hazy and there had

been a brown cloud that morning and it was extremely hazy that evening. The sun hadn't gone down yet, but you could look right at the sun and it wouldn't bother your eyes because it was being filtered by bad air.

Q. Not clouds?

A. No, it wasn't clouds. No, I wasn't legal to fly in clouds then. I now have my instrument rating so that I can land at Asheville on a clear, noncloudy day.

Q. Have you ever had to land with instruments?

A. Many times.

Q. During a bad air day?

A. Yes. Both here and San Diego.

MR. GULICK: I have no further questions, Your Honor.

MR. LANCASTER: We'll have no cross examination for this witness.

THE COURT: All right. That will complete your testimony and you may be excused.

(Witness stepped down.)

MR. GULICK: I do apologize, Your Honor, I do want to offer into evidence Dr. Russell's curriculum vitae which is Exhibit Number 41 -- excuse me, 441.

THE COURT: All right.

MR. GULICK: And for illustrative purposes only, Exhibits 213 and 214 which were the models of the bronchial

tubes.

And I'd also like to offer into evidence, Your Honor, the original -- this public record, the affidavit by George Carr of the Division of Medical Assistance with public records of expenditures by the Medicaid program for treatment of various diseases that are related to this case.

THE COURT: And that's Exhibit 45?

MR. GULICK: 45.

THE COURT: All right. Let those be admitted.

MR. GULICK: May I approach the clerk to hand her the original copy of this affidavit?

THE COURT: Yes.

(Plaintiff's Exhibits Numbers 45, 213, 214 and 441 were received into evidence.)

THE COURT: All right, gentlemen.

MR. GOODSTEIN: Your Honor, we're going to call as our next witness Dr. John Levy.

MR. LANCASTER: Your Honor, I have a preliminary matter I would like to raise about Dr. Levy.

THE COURT: All right.

MR. LANCASTER: We have filed two motions in limine that relate to Dr. Levy's testimony raising three separate grounds for exclusion of that testimony, and I think I can summarize it in about five minutes if I can be heard on it at this point.

THE COURT: Okay.

MR. GOODSTEIN: Your Honor, it was my understanding from the court that you were going to take these motions in limine in context during the trial, and we're prepared to lay a foundation with Dr. Levy with regard to his qualifications and his methodology. My suggestion is that we just go forward with the testimony; and at the appropriate time, if counsel wants to lodge an objection in context with the presentation of the evidence, then you can entertain it at that time. That would be our suggestion for how to proceed.

THE COURT: Okay. What is the witness's name?

MR. GOODSTEIN: Dr. John Levy, L-e-v-y.

THE COURT: All right. Now, what is the basis for your objection? Does it go to his qualifications as an expert?

MR. LANCASTER: No, sir. We have three separate bases for the objection.

One does go to the scientific reliability of his testimony, and the parties have favored the court with probably 50 or more pages of briefing on that issue. I could summarize it briefly, though.

THE COURT: Go ahead and let me hear you on that. Scientific basis --

MR. LANCASTER: Yes, sir.

THE COURT: -- for his testimony.

MR. LANCASTER: The scientific reliability issue, Your Honor, boils down to the fact that he has prepared a risk assessment which purports to calculate the exact number of mortalities that occur from small exposure changes in fine particulate matter air pollution. That kind of risk assessment has been presented to the United States Environmental Protection Agency in a standard setting context and the EPA has ruled it to be unreliable for purposes of setting an air quality standard, and so we question its scientific reliability.

Separately, Dr. Levy testified in his deposition that if he were to try to publish that kind of analysis in a peer reviewed journal, there are substantial uncertainties in the analysis and he would have to conduct an uncertainty analysis. He did not do that here and that is one of the hallmarks under the *Daubert* case from the Supreme Court that a witness should employ the same level of intellectual rigor in the courtroom that he does in his regular scientific practice.

Those are the two primary grounds of the unreliability point.

A second point, which is completely independent of that, is much simpler. What happened is, as the court has learned from the first several witnesses, Dr. Staudt specified an emissions projection for TVA for the year 2013 of sulfur dioxide at 450,000 tons per year. As he has acknowledged, he

has overstated that projection at least to the tune of 80,000 tons: The tons that will not be emitted from the Bull Run plant and the Kingston plant near North Carolina as a result of the scrubbers he indicated would be going on. And he handed that information to Drs. Chinkin -- or Messrs. Chinkin and Wheeler who explained how they ran it through their computer models. And they handed their output to Dr. Levy and he used that to calculate the mortalities.

So he has used as the basis for his testimony information -- numerical information about emissions and impacts that is contrary to the evidence in the record based on Dr. Staudt's testimony. And under the Fourth Circuit's *Tiger Construction* case holding that expert opinion evidence based on assumptions not supported by the record should be excluded, we seek exclusion on that basis.

The third ground is much narrower and would not result in the complete exclusion of Dr. Levy's testimony, but rather, merely the narrowing of it.

Dr. Levy has calculated state by state the number of people he believes are killed by what the plaintiffs call TVA's excessive emissions each year and has calculated, for instance, 49 deaths in New York, 60 in Florida, 62 in Indiana, 74 in Illinois, 110 in Ohio, as far as Minnesota, Massachusetts, Texas, Kansas. And consistent with the court's ruling this morning and consistent with the court's ruling on

one of TVA's motions about the preclusive effect of Alabama's statutory preemption, this case should be about air pollution in North Carolina.

And so if Dr. Staudt -- excuse me, if Dr. Levy does testify, we would ask the court to limit his testimony to his opinion about deaths caused in North Carolina and not in far away places like Ohio and Michigan and New York.

Those are the three grounds that we have laid out for objecting to Dr. Levy's testimony.

MR. GOODSTEIN: Your Honor, TVA is going to have an opportunity to present their case. This is North Carolina's opportunity to present its case. We have a qualified expert who's going to present relevant and reliable testimony. That's the standard under Rule 702, as you're well aware, Your Honor. We have responded to this motion in limine at length. The papers are in. Our motion in limine response is trial Exhibit 459. It's an extensive response, Your Honor. It shows that there's a very sufficient and reliable scientific basis.

This is just part of the response. And these are a select number of peer-reviewed journal articles in 459, Exhibit 2 attached to Dr. Levy's declaration, that show that the Health Impact Assessment Methodology that he's employed with Dr. Spengler supporting their opinions in reaching their conclusions in this case has been extensively published in the

peer-reviewed literature. It's been used by EPA. It's been used by a number of states. It's been tested. It's been subject to peer review. It was used by the U.S. Environmental Protection Agency in support of their Clean Air Interstate Rule. It's been subject to extensive publication. There are a number of peer-reviewed journal articles that document it in our response. It has a known error rate, and that's discussed in Dr. Levy and Dr. Spengler's report. It has standards to control it, which Dr. Levy and Dr. Spengler have documented in their report. And it's been generally accepted, as I've said, by EPA, governmental agencies and scientists in the field of epidemiology and public health. And this is merely a presentation of the expert testimony that TVA is going to present in its case. And really, the sole basis for this motion in limine are several affidavits from their witnesses. So this is really just a presentation of their case by -- in view of a motion in limine. There's really no question about the scientific basis of this testimony. And nobody can really say, after hearing the foundation that we're prepared to lay for this, that there's no scientific basis for the testimony.

THE COURT: All right. Let me have a few minutes here to review some notes on this.

MR. GOODSTEIN: Thank you, Your Honor.

(Pause.)

THE COURT: All right. We will proceed with the

testimony and if you will enter your objections as we get to the objectionable part, then I will rule on it. I want to see what sort of foundation the state proposes to lay before it tenders the various exhibits into evidence and I'll rule on them one by one and note your objections.

MR. GOODSTEIN: Thank you, Your Honor.

MR. LANCASTER: Mr. Goodstein addressed the scientific reliability issue that the court appeared to review. The separate issue was the scope of the opinions, and our motion is consistent with what I understood the court to say earlier today; that Dr. Levy is going to attempt to calculate numbers of deaths caused by what plaintiff calls TVA's excessive pollution. These deaths occurring in Pennsylvania, Ohio, Indiana, Illinois, Michigan and far away states --

THE COURT: And I won't accept that evidence, as I indicated. I'm interested in these three states primarily and I'll allow the gentleman to state his opinion, but we're not going into details, as I indicated, on these other matters.

MR. LANCASTER: Thank you, Your Honor.

THE COURT: That's not relevant here.

MR. GOODSTEIN: Your Honor, since day one in this lawsuit, North Carolina has alleged that not only are there these types of impacts occurring in North Carolina, but that they're occurring throughout the region. And this is, as

stated in our complaint, North Carolina pled this case as having to do with regional air pollution impacts and this is a very important issue for this case, obviously.

THE COURT: All right. Go ahead with your evidence and I'll hear you at the appropriate time.

MR. GOODSTEIN: I appreciate it, Your Honor. Thank you very much.

THE COURT: Call your witness.

THE COURT: Dr. Levy.

JONATHAN IAN LEVY,

being first duly sworn, was examined and testified as follows:

MR. GOODSTEIN: Your Honor, if I may approach. We have a set of exhibits for Dr. Levy.

THE COURT: Yes.

DIRECT EXAMINATION

BY MR. GOODSTEIN:

Q. Good afternoon, Dr. Levy.

A. Good afternoon.

Q. Can you state your full name for the record, please.

A. Jonathan Ian Levy.

Q. And how are you currently employed, Dr. Levy?

A. I'm an associate professor of Environmental Health and Risk Assessment at the Harvard School of Public Health.

Q. You should have in front of you a book of exhibits. And the first one should be Plaintiff's Exhibit 429 for

identification. Is that a copy of your CV?
A. Yes, it is.
Q. And can you tell us what your responsibilities are in your current position.
A. Well, I am on the faculty in the Department of Environmental Health at Harvard School of Public Health, and that's a position that involves a combination of research and teaching. So I'm involved in research on air pollution exposures and health effects called Air Pollution Risk Assessment. And I also teach courses. I teach the course, the introductory course on risk assessment to master's and doctoral students in our program. I also teach courses on research methods in general in environmental health, and I teach a freshman seminar at Harvard College on urban environmental health.
Q. And do you have any updates to your CV, Plaintiff's Exhibit 429?
A. There certainly are some updates. This CV was circa October 2006. There's a number of additional publications. Many publications that are listed in the CV as submitted are now published and there are additional publications that have now been submitted.
I guess also, I'm listed in terms of my affiliations as a member of the Society for Risk Analysis. I remain a member, but I'm now the president of the Society for Risk Analysis'

New England Chapter. So that is an update.

And certainly updates to the number of students that I have mentored and their status. Some students, thankfully for them, have graduated since October 2006.

Q. And what departments are you associated with at the Harvard School of Public Health?

A. My primary appointment is in the Department of Environmental Health and it's within a program called the Exposure Epidemiology and Risk Program. And this is a program that involves multi-disciplinary and inner-disciplinary research on exposures on epidemiology and on risk assessment generally related to air pollution but including other stressors as well.

And I also have a secondary appointment in the area of health policy and management which generally involves a number of things, but my affiliation is related to the application of decision theory methods and other methods for policy analysis.

Q. And can you describe each of these departments, Department of Environmental Health and the Health Policy and Management Department, please.

A. The Department of Environmental Health is a large department. There are a number of faculty members who conduct research in environmental epidemiology, in characterizing human exposures to a variety of pollutants, conducting risk assessment, as well as laboratory-based studies looking at

human physiology, looking at exposures of animals to different pollutants and understanding health effects.

And health policy and management is a large crosscutting department that includes aspects of healthcare systems and financing. For example, my arm of research is more on policy analysis methods, and there's a number of faculty there who are involved in quantitative methods for policy analysis.

Q. And what courses do you teach at the Harvard School of Public Health?

A. As mentioned, I teach a course on risk assessment. This is an introductory course for students in our program, for master's students and doctoral students in which we go through the elements of a risk assessment and then students conduct risk assessments themselves as part of the case base structure of the course.

The other class at the School of Public Health that I alluded to, which is a research practicum class, involves teaching second year master's students how to conduct a research study, what is involved, how to develop hypotheses, how to analyze data and write up papers.

Q. And what other courses have you taught?

A. I've been involved in instructing a number of different courses. I've contributed lectures, for example, in the introductory course for master of public health students on risk assessment methods. So that has happened over a number

of years.

I've also lectured in our air pollution course on exposure assessment methods and modeling.

And I have taught in a series of freshman seminars at the college currently about urban environmental health, but in the past related to issues of environmental equity or broadly health and inequality.

Q. And you're also a research adviser?

A. Yes, that's correct. I advise some master's students, but I largely advise doctoral students. I have had three of my doctoral students who have graduated and gone forth into the world and I currently advise another five doctoral students, and I have served or am serving on the dissertation committees for perhaps another dozen students.

Q. And you do some consulting in environmental risk as well as your research duties?

A. I do a small amount of consulting. I don't have anything that would resemble a formal practice, but there have been opportunities such as this one in which my academic research can be extended and applied in other areas, and so I have taken on a small number of projects over the years.

Q. Can you tell us what a few of those projects have been and are related to your testimony in this case.

A. Sure. I prepared a report for the City of Alexandria, Virginia, about health impacts of some of the local power

plants which was an expansion of a publication that is listed in my curriculum vitae looking at five power plants in the Washington, D.C. area.

I also prepared a report for the Maryland Nurses Association, again, an extension of this Washington, D.C. analysis, looking at a few other power plants in Maryland specifically and trying to understand and quantify the health impacts from those plants.

I also participated in an administrative hearing in Wisconsin regarding a power plant there in which Dr. Spengler and I and others prepared an expert report regarding the health impacts of emissions from a power plant in Wisconsin under alternative control scenarios.

Q. And you testified as an expert in that proceeding?

A. Yes, I did.

Q. And what was the subject matter of your testimony there?

A. It was related to the application of health impact assessment methods for air pollution and the quantification of health impacts from power plant emissions.

Q. So it was a similar methodology to the methodology that you've employed in this case?

A. Very similar, yes.

Q. And what were you asked to do in this case by the attorney general of North Carolina?

A. I was asked to prepare an air pollution health impact

assessment very similar to the sort that I've done over more than a decade related to power plant emissions as well as other source emissions in the peer-reviewed academic literature and so asked to conduct a health impact assessment focusing on what's been termed the excess emissions of the TVA power plants and quantifying the effects of those emissions on public health.

Q. All right. Can you summarize for us your educational background, Dr. Levy. And this is on Page 1 of Plaintiff's Exhibit 429.

A. Sure. I have an undergraduate degree from Harvard College in applied mathematics. And then after working for a few years, I went back to graduate school and I obtained my Doctorate in Environmental Science and Risk Management which is a joint degree program between environmental health and health policy and management, the two departments with which I'm currently affiliated. And that involved, obviously, course work and then a dissertation that was completed in 1999 building a health impact assessment model for power plants. And so that -- you can see the title of the dissertation is Environmental Health Effects of Energy Use: A Damage Function Approach. And damage function is another term for health impact assessment as I have described it.

I then had a year and a half of post-doctoral training also at the Harvard School of Public Health. I should have

said my doctoral degree is from the Harvard School of Public Health. I did a year and a half of post-doctoral training both in environmental health and in biostatistics working with faculty in both departments.

And then I joined the faculty at the School of Public Health in February of 2001.

Q. You mentioned that your doctoral dissertation at Harvard School of Public Heath dealt with health issues associated with power production. Can you give us a little more background about that particular dissertation.

A. Sure. It involved trying to develop the tools and techniques to quantify the health impacts from emissions from, in this case, one selected power plant. And so the work had a number of different components, two of which ended up published in the peer-reviewed literature. One was a meta-analysis or a statistical review of the particulate matter mortality literature available at that time which we used to develop what we call a concentration response function looking at the relationship between particulate matter exposure and the risk of mortality, and so that function was developed as part of the dissertation work and published in the year 2000.

And then I applied that function as well as functions for a number of other health outcomes for particulate matter and for ozone. I also applied an atmospheric dispersion model and

then combined the evidence using standard health impact assessment methods to quantify the health impact associated with emissions from one power plant in Massachusetts.

Q. And how does that method that you developed in your dissertation compare to the method that you used in this case to develop your conclusions?

A. It's a very similar methodology in general. The atmospheric dispersion model conducted by Sonoma is far more advanced, I will admit, than what I conducted in the late '90s since the computer modeling has gotten much better over time. The epidemiologic evidence and other evidence about health effects of particulate matter and ozone has grown exponentially in the intervening years but the core methodology has not changed.

Q. And did that lead to some publications in the peer-reviewed literature?

A. The dissertation work specifically, yes, led to two publications, one in 1999 in the Journal of Environmental Science and Technology and another in 2000 in the Journal of Environmental Health Perspectives.

With that foundation that we laid and that I developed in my dissertation, I then did a series of follow-on investigations in subsequent years starting as a post-doctoral researcher and then extending into my faculty position in which we applied health impact assessment methods in four

separate publications looking at two power plants in Massachusetts, looking at, I believe it was nine power plants in Illinois, five power plants in the Washington, D.C., area and I believe seven power plants in Georgia. And those led to four separate publications all involving the application of the health impact assessment methods.

Q. Can you please, Dr. Levy, direct us to those publications in your CV.

A. Sure. That would begin on page, what is labeled Page 9 of 14 on my CV, and which is where the bibliography begins. And so the two publications that I mentioned that were tied to my dissertation work, the first one is Number 6, Development of a New Damage Function Model for Power Plants: Methodology and Applications. Publication Number 8 is the other publication that I --

Q. Just give me one second.

A. Yes.

MR. GOODSTEIN: We're on Plaintiff's Exhibit 429, Your Honor. Should be the first one in the binder from Dr. Levy.

THE COURT: All right.

Q. Can you show us where that is again.

A. Sure. As mentioned, the first of the publications from the dissertation work is reference Number 6 on that page which is now highlighted which is in the journal Environmental

Science and Technology in 1999.

The paper in which we developed the concentration response function for particulate matter is reference Number 8 from the journal Environmental Health Perspectives.

Q. And these are all published in peer-reviewed journals?

A. That's correct. These are all peer-reviewed journals on this CV.

And then I alluded to the four additional power plant applications that we'd had in the past. The first one of those begins on the following page on Page 10 at the top of the page. That's reference Number 13, Modeling the Benefits of Power Plant Emission Controls in Massachusetts, a paper that Dr. Spengler and I wrote.

The one immediately below it, reference Number 14, was the study that I alluded to in Illinois quantifying the impacts of nine power plants there.

Scan down. Reference Number 20 is the third of the four power plant health impact assessment case studies. This was looking at power plants near Washington, D.C., as the title alludes to.

And the final publication is the last publication on that same page, Reference 27, looking at power plants in Georgia. The term intake fractions in the title refers to exposure to the population from those emissions. So this paper was focusing on modeling the exposure of the population from those

power plants.
Q. All right. How did the methodologies that were documented in those peer review journal articles compare to the methodology that you used in this case?
A. It was very similar to what was applied in this case. The details differ slightly depending on the timing of the paper or the focus of the paper. For example, the reference number 20 of the paper focusing on Washington, D.C. power plants was aimed at understanding which susceptible subpopulations may be at greatest risk and how the risk was distributed. So that involved some different methods and different assumptions than other applications. But the broad brush methodology in all of these peer-reviewed publications is the same as what we conducted here.
Q. Can you briefly describe for us your professional background, Dr. Levy.
A. Sure. Other than my current position as a faculty member of the School of Public Health, as mentioned, I did work in the intervening years between undergraduate and graduate school. This was at an economic and management consulting firm just outside of Boston in which I developed computer simulation models for Fortune 500 companies doing things like forecasting sales and trying to project the impact of different advertising campaigns on sales of products. So not within the subject matter here, but certainly involving

development of computer simulation models.
Q. And you did some post-doctoral training in statistical analysis.
A. That's correct. As mentioned, this was -- my post-doctoral training was jointly between environmental health and biostatistics and so part of my post-doctoral time involved these health impact assessment studies, but I also worked with faculty in biostatistics on models to quantify exposure heterogeneity in urban environments trying to develop techniques to understand whether and why air pollution might be different from one road to another depending on proximity to a large bus terminal, for example.
Q. You went back to the School of Public Health in 2001 and what was your position at that time?
A. I was hired as an assistant professor at that time and served as an assistant professor until 2006 when I was promoted to associate professor within the department.
Q. And what did that promotion involve?
A. Well, it broadly involves review of my curriculum vitae, my publications, my teaching record. I believe that some of the criteria are having a national or international reputation and work that has been, you know, well accepted within the scientific community. The senior faculty in the department could probably speak more to the details of the promotion process than I could. But it involves a formalized review of

the academic productivity as well as the teaching record.
Q. And what are your academic appointments in environmental health?
A. I'm not sure I understand the question.
Q. What are your positions? What are your academic positions?
A. In terms of my responsibilities?
Q. Yes.
A. As an associate professor in the department, I certainly conduct research and that's -- you know, one of the primary things that a faculty member at the School of Public Health does is write grants, conduct primary research. So I'm the principal investigator of a number of grants, perhaps 15 or so over the years that I've been on the faculty. You know, maybe, I guess, four grants currently that I serve as principal investigator of. So that is a primary line of activity. Conducting research and then supervising graduate students on those research products.

I'm also involved in teaching and I serve on a number of school committees as well, including committees on curriculum development within the department. I serve on the faculty council of the school which is an elected position involving some oversight or at least nominal oversight of school activities.

And I'm also an appointed member on the committee on

concerns of women faculty which is related to gender balance and gender inequity within the school and the university as a whole.

Q. And one of your research grants is listed on Page 1 of your CV under Honors and Awards; is that right?

A. That's correct. This was the -- it's the first honor and award listed. The Health Effects Institute, Walter Rosenblith New Investigator Award. And this is an award/grant that is -- it's a competitive award process in which new faculty members, new researchers apply and are evaluated by the Health Effects Institute which is a nonprofit entity jointly funded by EPA and by industry, largely the automotive industry. As I said, it's a competitive process and I was excited to receive that award which provided both the honor of the award and then a grant that I've been conducting over the past three years.

Q. And can you tell us what that research involves, please.

A. That project is focused on spatial patterns and spatial and temporal patterns of air pollution exposures in the Boston area looking at both indoor and outdoor environments, focused largely on $PM_{2.5}$ or fine particulate matter, but also looking at other air pollutants. And this is part of an epidemiologic study nested within an epidemiologic study looking at risk factors for asthma in the urban environment.

Q. And you are currently doing that research now?

A. Yeah, that is an ongoing project.

Q. Can you please describe for us some of your other primary current research projects.

A. Sure. Two other major research projects I can highlight -- there's always a large number of projects going in and out at any point in time.

I am involved in a large scale collaborative funded by the Federal Aviation Administration looking at exposures to air pollution, health risks from air pollution associated with the aviation industry. This is a multi-university and industry collaborative. MIT is the head of this organization and we work along with MIT and the University of North Carolina on some of our projects which involve health impact assessment methods as applied to the aviation industry using CMAQ as an atmospheric dispersion model and then quantifying the air toxic and critera pollutant health impacts.

And we've also done a series of monitoring studies around airports to then quantify the contribution of emissions from the airport to concentrations in the nearby neighborhoods.

Another ongoing study is related to air pollution within New York City focusing on contributors to spatial gradients in New York City trying to model patterns of air pollution, understand the implications of being closer to or further from major roads, street canyons, and the like, and then ultimately involving the application of health risk assessment methods to understand the benefits of traffic mitigation measures in New

York City.

Q. And can you describe some of your previous research projects that are directly pertinent to this matter.

A. Sure. Probably the most pertinent ones I alluded to earlier, the sequence of four power plant health impact assessment studies, and those were all funded within a single grant so I won't belabor the details of those.

I was also funded by the U.S. Environmental Protection Agency to conduct a meta-analysis of the ozone mortality literature. EPA was obviously interested in what the relationship was between ozone and mortality and so they found three different research groups that they asked to independently look at the question, not talk to each other in the process; and then upon completion of the analyses, we published the three papers jointly.

And so I was one of the three groups involved in that. There was another group from Yale and Johns Hopkins and a third group from NYU that was involved in the study.

And so that culminated in a publication in which we synthesized the literature, the epidemiologic literature on ozone mortality and developed a concentration response function. That's the function that we ended up using within this health impact assessment.

Q. What is a concentration response function?

A. That is a quantitative relationship between a level of

pollution in the ambient environment, we're talking about concentrations, and the frequency of response. And response could be premature death. It could be hospital admission. It could be asthma attack. So it can be any of the health outcomes that can be associated with pollution. So it is formally quantifying that relationship generally for later application in a health impact assessment.

Q. And how do you develop a concentration response function?

A. What we generally do is pull together all the publications that are relating the exposure and the outcome. We read them critically and generally include a subset that meet standards that we develop in advance, and then the studies are pooled quantitatively taking account, for example, of their statistical strength so that studies with more uncertainty may get less weight and studies with less uncertainty would get more weight. And so we use quantitative methods to pool those studies to come up with essentially the collective quantitative wisdom from the body of evidence.

Q. Have you published a number of papers in the peer-reviewed journal -- in peer review journals deriving concentration response functions?

A. Yes. There was the earlier paper I alluded to for particulate matter mortality from the year 2000 that we developed. And then the publication in the year 2005 which I can point out on the CV if that is helpful.

Q. Please do. That's Plaintiff's Exhibit 429.
A. Sure. And this would be on page 11 of 14, and reference number 31. So this -- this is the paper that I alluded to earlier, the study that was funded by USEPA to synthesize the ozone mortality literature and develop a concentration response function.

We then had a follow-on paper -- or I should say I did -- it didn't involve the same coauthors -- in which we used this function and then developed other functions and applied them to a health impact assessment in California. That would be the last publication on that same page on Page 11, reference Number 41, the Health Benefits of Reduced Tropospheric Ozone in California. And so that study involved both the quantification of health benefits, but also the development of concentration response functions.

Q. I'm sorry, what page was that on?
A. That's on that same page. On Page 11. It's highlighted now on the screen.
Q. Okay. So it's Peer Review Publication 41.
A. Yes, that's correct.

And then I should say I was involved in an earlier study examining the ozone literature on both mortality and morbidity or nonfatal effects and that was -- we have to go back a few pages for that. That's on Page 9 of 14, the very last reference on that page, reference Number 12, Assessing the

Public Health Benefits of Reduced Ozone Concentrations. And so that was an earlier study. And the first study -- or I should say the first study following the 1999 publication in which we synthesized the ozone literature and developed concentration response functions.

And there's a significant amount of new literature in this area that comes out each year. It probably has literally grown exponentially over the last ten years. I think subsequent to the $PM_{2.5}$ standard being promulgated and subsequent to certainly potential changes and actually now changes in the ozone National Ambient Air Quality Standards, I've seen some indication that the number of publications just on fine particulate matter cardiovascular effects is somewhere in the order of two to three hundred publications in a year, and that may be a conservative estimate. So it's a massive literature that is growing by leaps and bounds every year.

Q. So the concentration response function that you've developed, does that develop over time into literature?

A. Oh, sure. I mean, we would not want to use functions developed in 1997 today. The literature evolves. It changes. There's new publications. There's new understandings. So in each of these health impact assessments and in each of these applications we look at the literature anew. We look for new studies and we reintegrate and resynthesize the literature. So the numeric values can change. The health outcomes

included or excluded can change. The general approach does stay the same, though.

Q. And Dr. Levy, do you serve on a number of advisory boards, committees and commissions related to epidemiology and exposure to air pollutants?

A. That's correct. Probably the two most significant ones perhaps in this contest are two committees with the National Academy of Science, the National Research Council branch or the NRC. They assemble a number of ad hoc expert committees to look at specific subject areas and then to write reports that provide recommendations to whoever had requested the study, whether it be Congress or EPA or others.

So I served on one committee from 2004 to 2006 which was focused on changes to the new source review program and trying to understand the possible implications for air quality and public health.

I'm currently serving on a National Research Council committee involving examining the field of risk assessment as a whole and trying to consider what areas may require changes and what areas are working well. The field of risk assessment has been around for a while, but the seminal report that really developed it was a 1983 report by the National Research Council, and so this expert committee was assembled to prepare a report in honor of the 25th anniversary of that original report to basically see what's happened in the world and in

the field in the last 25 years and what could happen or what should happen over the next 25 years. And that committee is still ongoing. We have not yet issued our report.

Q. All right. And you also mentioned your involvement in the Society for Risk Analysis.

A. That's correct. I've been a member of the Society of Risk Analysis since I was a graduate student. Certainly attend their annual meetings. And I'm currently serving as the president of the New England Chapter of the Society of Risk Analysis.

Q. And can you tell us what the field involves, the field of environmental risk analysis.

A. Well, risk analysis is a very broad area that includes both the analytical and assessment phase and then a lot of other pieces such as risk communication. Risk assessment involves, you know, as defined by the National Research Council in 1983, a sort of codified to include four key steps, really, known as the four steps established in this report which is referred to in the field as the Red Book because the title is long and the report has a red cover. So that's the shorthand and I may lapse into that shorthand from time to time. But the Red Book in 1983 really laid out the foundation for the field. And if it would be useful, I could try to sort of sketch those steps out or I can speak to it in broader brush terms at this point.

Q. If we can, I want to try and complete your background and experience in environmental risk analysis.
A. Okay. So I mean, I'll just say it broadly. Risk assessment involves the combination of exposure assessment and dose response assessment, you know, which is similar to concentration response, you know, looking at the relationship between exposures and health outcomes and synthesizing that evidence to yield quantitative estimates of health risks.
Q. And you have a number of publications in that field listed in your CV. We've looked at some of them. Can you summarize your peer-reviewed publications in the field of environmental risk analysis.
A. Beyond those that were mentioned, I've been involved in other applications looking at, for example, life cycle analysis methods and how one can introduce risk assessment concepts into the field of life cycle analysis which is basically looking at product or processes from cradle to grave, from manufacture to disposal. So we've tried to develop risk assessment methods applicable there.
A number of the studies that I've alluded to, a number of ongoing studies focused on the impact of aviation or the impact of traffic. So I could walk through the bibliography, but it is probably fair to say that a majority of the listed publications in my CV are either direct risk assessment applications, methodological risk assessment developments or

publications such as the ones that I mentioned on concentration response functions that try to build up a component of a risk assessment.

Q. And do you currently have an article that's been accepted for publication by the Risk Assessment Journal?

A. I have two articles, actually, that are currently in press in the Journal of Risk Analysis.

Q. And is that the journal that Dr. Moolgavkar is a reviewer for?

A. He's actually, I believe, an area editor or an associate editor of the journal.

Q. And was he one of the primary reviewers of that article that you submitted and what has been accepted for publication?

A. He wasn't the primary reviewer, but he was the area editor involved which means he, you know, was involved in some potential triaging of articles. Some articles don't get out to the review process. You know, this article did get out to the review process and then, you know, he presumably was involved in reading the reviews and coming to the decision for the journal about whether the article was or was not acceptable.

Q. And have you written a number of book chapters in environmental risk analysis?

A. I have. Probably the most pertinent ones are -- were in case studies in China and in Mexico City, in each case trying

to both develop concentration response functions and to quantify health impacts.

In China this was a book, I believe, out of MIT press in collaboration with Mario Molina who has since left MIT, but the noble laureate from Mexico who first identified the hole in the ozone layer. So we worked in collaboration with his group to apply health impact assessment methods to Mexico City, understand the health implications of different pollutants, and that culminated in a book.

And in addition, within China to look at the health impacts of air pollution across different sectors of the Chinese economy.

Q. And are you a peer reviewer on journals and other organizations that publish reports in the area of environmental health analysis?

A. I serve as a reviewer for a large number of journals. The full list is listed in my CV. Maybe 20 some odd journals, including the Journal of Risk Analysis itself as well as other journals like Environmental Science and Technology or Environmental Health Perspectives in which a lot of risk assessment work is published.

I've also served as a peer reviewer to the National Research Council on some of their expert reports, including one, a 2002 expert report on health benefits analysis methods which is yet another name for health impact assessment as

applied to air pollution.

I also served on a -- as a peer reviewer of the recent ozone mortality National Research Council report that came out in 2008 that I believe was alluded to sometime this morning.

I've also served as a peer reviewer for a number of granting agencies like the USEPA, the National Science Foundation and others.

And served as both a peer reviewer and scientific adviser to the California Air Resources Board as they have tried to understand the health impacts of both ozone and fine particulate matter within some of their regulatory impact analyses or health impact assessments.

Q. And these peer reviewer activities are listed on Page 3 of your CV; is that right? Plaintiff's Exhibit 429.

A. That's correct. It's in the top area of Page 3 under the heading Peer Reviewer.

Q. And that's on your screen right now.

A. That's correct.

Q. Can you also summarize for us, Dr. Levy, your prior testimony in environmental risk analysis.

A. Sure. I've been asked to present on either the health impacts of fine particulate matter or specifically on the impacts of power plant pollution on public health, looking at the quantitative risk assessment side of things. I've testified on a couple of occasions before the U.S. Senate

before their Environment and Public Works Committee. And I've also testified in a number of state agency hearings either before legislative bodies or in other public hearings, that included in Massachusetts, Connecticut, New Hampshire and Illinois.

MR. GOODSTEIN: Your Honor, at this time we tender Dr. Levy as an expert in environmental risk analysis.

MR. LANCASTER: And we have not had any objection to his qualification in environmental risk analysis as he defined it. Our objection went to the scientific reliability under *Daubert*.

I've been thinking about it as he's been testifying, if I could be so bold as to make a suggestion.

The case law recognizes that in a bench trial, rather than a jury trial, it's acceptable for the court to receive the evidence subject to an objection under *Daubert versus Merrell Dow Pharmaceuticals* and then rule on the evidence's admissibility after hearing it. There's not the danger of exposing a jury inadvertently to inadmissible testimony since it's not a jury trial.

My suggestion would be that the testimony be taken in terms of the scientific reliability subject to the objections that we explained in court documents 108, 108-2 and 127. I realize they are lengthy briefings. They are lengthy explanations. And although plaintiff's counsel indicated that

nobody could question the scientific reliability, I must be that nobody, and we've explained that thoroughly in our briefing.

MR. GOODSTEIN: Your Honor, this is the best scientific evidence available and there's no reason why this court needs to receive this evidence subject to any reservations whatsoever. And we've laid the foundation for this in the reports that we've submitted on behalf of these experts during discovery. We've laid the foundation in their depositions. And we're prepared to lay the foundation here at trial if the defendant persists in this, what we think is really a just meritless argument that there is no scientific basis for this testimony.

And we will now move on now that Dr. Levy has been tendered and received as an expert, we'll move on to laying the foundation for his methodology that he used in this case, Your Honor, and it will be very clear from the record once we lay this out that there is very substantial peer-reviewed scientific bases for his testimony and his methodology.

So there's really no question about this. We've laid it out extensively in our response to their motion in limine that's marked as trial Exhibit 459 in our books, Your Honor. We have an affidavit from Dr. Levy as part of those materials. This is the select amount of scientific evidence that I have here in my hand that is attached to Dr. Levy's

declaration. These are all publications in peer-reviewed journals not only by Dr. Levy, but by a number of eminent scientists around the country that have used the health impact assessment methodology to evaluate health impacts from air pollution, from power plants and other similar studies.

So there's really -- there won't be any question. We can do it any way the court would like. We've done it in our papers, with declarations and legal memorandum. We can do it here with Dr. Levy. It's important testimony for you to consider, Your Honor, because it also goes to the weight of his conclusions. And it's going to take a few more minutes, but it's important to lay out the methodology so you can consider his conclusions and give them the weight that is appropriate given the vast amount of scientific evidence that supports it.

THE COURT: Well, I'm going to find that he is as tendered an expert in this field of risk analysis and subject matter on which he proposes to testify here, epidemiology. And he obviously has published numerous articles for peer review. He's presently a peer reviewer, if I understood his testimony. He has conducted substantial research and his methods have been adopted by other experts in the field. He's a leader in a large department, health department there at Harvard. Teaches in the field. Advises doctoral students. Consults on environmental risks, health impact. Has had

substantial involvement with prior analyses of health impact from power plant pollution emissions. His testimony is certainly relevant to the questions before the court. He's basing his opinions on scientific facts and data. He's shown a reliability of his methods. Proposes to apply these principles and methods to his analysis of the facts in this case. Proposes to testify to matters growing directly out of research that he personally conducted. His theories have been tested and subject to peer review and publication. He's developed rates of -- potential rates in the area. I think he's going to testify about that. If I'm understanding correctly, he says his techniques have achieved general acceptance in the relevant expert community.

It appears, at least at this stage, that he is qualified to go ahead with his testimony.

Now, as to the other testimony before the court, being a fact finder, in the unique position of being the fact finder in this case, I'm going to review all of this and, as with the other testimony, do an analysis of what is offered and consider the reliability of the testimony.

So I think what I'm saying to you -- both of you is that I'm agreeing in part with both of you and I'm going to have the last opportunity to review it and to -- under the *Daubert* and *Kumho Tire* and the electric company cases, come to a final decision as to whether or not the testimony is

admissible in part or in whole. And of course, I'll set that out in the final decision which I prepare in this case.

So we'll proceed at this point. And Mr. Lancaster, you go ahead and make your objections so they will be a part -- make your -- is my machine not working again?

MR. GOODSTEIN: We can hear you fine, Your Honor.

MR. LANCASTER: I'm a little hard of hearing, I'm sorry, Your Honor.

THE COURT: Let me speak into this mic a little more carefully, then.

You make your objections for the record so that when it's being reviewed on appeal, as it most likely will be, then if the court's -- well, not if, the court will need to review what happens here and be aware that your objections were timely made. So don't feel uncomfortable about making them.

MR. LANCASTER: Thank you, Your Honor.

MR. GOODSTEIN: Thank you, Your Honor.

THE COURT: All right. Let's -- it's almost 6 o'clock.

MR. GOODSTEIN: Yes, sir.

THE COURT: Well, we might as well start him fresh in the morning.

MR. GOODSTEIN: Very well, Your Honor. That's always a good plan.

THE COURT: Okay. Take a recess until tomorrow

morning at 9 o'clock.

(Evening recess at 5:53 p.m.)

UNITED STATES DISTRICT COURT

WESTERN DISTRICT OF NORTH CAROLINA

CERTIFICATE OF REPORTER

I certify that the foregoing transcript is a true and correct transcript from the record of proceedings in the above-entitled matter.

Dated this 17th day of July, 2008.

s/Cheryl A. Nuccio
Cheryl A. Nuccio, RMR-CRR
Official Court Reporter